

195 Human Secreted Proteins

Related Applications

- 5 This application is a continuation-in-part of PCT/US02/08279, filed March 19, 2002, which in turn claims benefit of the following:

Application::	Continuity Type::	Parent Application::	Parent Filing Date::
PCT/US02/08279	Continuation-in-part of	10/100,683	03/19/02
10/100,683	Non-provisional of	60/277,340	03/21/01
10/100,683	Non-provisional of	60/306,171	07/19/01
10/100,683	Non-provisional of	60/331,287	11/13/01
10/100,683	Continuation-in-part of	09/981,876	10/19/01
09/981,876	Divisional of	09/621,011	07/20/00
09/621,011	Continuation of	09/148,545	09/04/98
09/148,545	Continuation-in-part of	PCT/US98/04482	03/06/98
10/100,683	Continuation-in-part of	09/621,011	07/20/00
09/621,011	Continuation of	09/148,545	09/04/98
09/148,545	Continuation-in-part of	PCT/US98/04482	03/06/98
10/100,683	Continuation-in-part of	09/148,545	09/04/98
09/148,545	Continuation-in-part of	PCT/US98/04482	03/06/98
10/100,683	Continuation-in-part of	PCT/US98/04482	03/06/98
PCT/US98/04482	Non-provisional of	60/040,162	03/07/97
PCT/US98/04482	Non-provisional of	60/040,333	03/07/97
PCT/US98/04482	Non-provisional of	60/038,621	03/07/97
PCT/US98/04482	Non-provisional of	60/040,161	03/07/97
PCT/US98/04482	Non-provisional of	60/040,626	03/07/97
PCT/US98/04482	Non-provisional of	60/040,334	03/07/97
PCT/US98/04482	Non-provisional of	60/040,336	03/07/97
PCT/US98/04482	Non-provisional of	60/040,163	03/07/97
PCT/US98/04482	Non-provisional of	60/047,615	05/23/97
PCT/US98/04482	Non-provisional of	60/047,600	05/23/97
PCT/US98/04482	Non-provisional of	60/047,597	05/23/97
PCT/US98/04482	Non-provisional of	60/047,502	05/23/97
PCT/US98/04482	Non-provisional of	60/047,633	05/23/97
PCT/US98/04482	Non-provisional of	60/047,583	05/23/97
PCT/US98/04482	Non-provisional of	60/047,617	05/23/97
PCT/US98/04482	Non-provisional of	60/047,618	05/23/97
PCT/US98/04482	Non-provisional of	60/047,503	05/23/97
PCT/US98/04482	Non-provisional of	60/047,592	05/23/97
PCT/US98/04482	Non-provisional of	60/047,581	05/23/97
PCT/US98/04482	Non-provisional of	60/047,584	05/23/97
PCT/US98/04482	Non-provisional of	60/047,500	05/23/97
PCT/US98/04482	Non-provisional of	60/047,587	05/23/97
PCT/US98/04482	Non-provisional of	60/047,492	05/23/97

PCT/US98/04482	Non-provisional of	60/047,598	05/23/97
PCT/US98/04482	Non-provisional of	60/047,613	05/23/97
PCT/US98/04482	Non-provisional of	60/047,582	05/23/97
PCT/US98/04482	Non-provisional of	60/047,596	05/23/97
PCT/US98/04482	Non-provisional of	60/047,612	05/23/97
PCT/US98/04482	Non-provisional of	60/047,632	05/23/97
PCT/US98/04482	Non-provisional of	60/047,601	05/23/97
PCT/US98/04482	Non-provisional of	60/043,580	04/11/97
PCT/US98/04482	Non-provisional of	60/043,568	04/11/97
PCT/US98/04482	Non-provisional of	60/043,314	04/11/97
PCT/US98/04482	Non-provisional of	60/043,569	04/11/97
PCT/US98/04482	Non-provisional of	60/043,311	04/11/97
PCT/US98/04482	Non-provisional of	60/043,671	04/11/97
PCT/US98/04482	Non-provisional of	60/043,674	04/11/97
PCT/US98/04482	Non-provisional of	60/043,669	04/11/97
PCT/US98/04482	Non-provisional of	60/043,312	04/11/97
PCT/US98/04482	Non-provisional of	60/043,313	04/11/97
PCT/US98/04482	Non-provisional of	60/043,672	04/11/97
PCT/US98/04482	Non-provisional of	60/043,315	04/11/97
PCT/US98/04482	Non-provisional of	60/048,974	06/06/97
PCT/US98/04482	Non-provisional of	60/056,886	08/22/97
PCT/US98/04482	Non-provisional of	60/056,877	08/22/97
PCT/US98/04482	Non-provisional of	60/056,889	08/22/97
PCT/US98/04482	Non-provisional of	60/056,893	08/22/97
PCT/US98/04482	Non-provisional of	60/056,630	08/22/97
PCT/US98/04482	Non-provisional of	60/056,878	08/22/97
PCT/US98/04482	Non-provisional of	60/056,662	08/22/97
PCT/US98/04482	Non-provisional of	60/056,872	08/22/97
PCT/US98/04482	Non-provisional of	60/056,882	08/22/97
PCT/US98/04482	Non-provisional of	60/056,637	08/22/97
PCT/US98/04482	Non-provisional of	60/056,903	08/22/97
PCT/US98/04482	Non-provisional of	60/056,888	08/22/97
PCT/US98/04482	Non-provisional of	60/056,879	08/22/97
PCT/US98/04482	Non-provisional of	60/056,880	08/22/97
PCT/US98/04482	Non-provisional of	60/056,894	08/22/97
PCT/US98/04482	Non-provisional of	60/056,911	08/22/97
PCT/US98/04482	Non-provisional of	60/056,636	08/22/97
PCT/US98/04482	Non-provisional of	60/056,874	08/22/97
PCT/US98/04482	Non-provisional of	60/056,910	08/22/97
PCT/US98/04482	Non-provisional of	60/056,864	08/22/97
PCT/US98/04482	Non-provisional of	60/056,631	08/22/97
PCT/US98/04482	Non-provisional of	60/056,845	08/22/97
PCT/US98/04482	Non-provisional of	60/056,892	08/22/97
PCT/US98/04482	Non-provisional of	60/047,595	05/23/97
PCT/US98/04482	Non-provisional of	60/057,761	09/05/97
PCT/US98/04482	Non-provisional of	60/047,599	05/23/97
PCT/US98/04482	Non-provisional of	60/047,588	05/23/97
PCT/US98/04482	Non-provisional of	60/047,585	05/23/97
PCT/US98/04482	Non-provisional of	60/047,586	05/23/97
PCT/US98/04482	Non-provisional of	60/047,590	05/23/97

PCT/US98/04482	Non-provisional of	60/047,594	05/23/97
PCT/US98/04482	Non-provisional of	60/047,589	05/23/97
PCT/US98/04482	Non-provisional of	60/047,593	05/23/97
PCT/US98/04482	Non-provisional of	60/047,614	05/23/97
PCT/US98/04482	Non-provisional of	60/043,578	04/11/97
PCT/US98/04482	Non-provisional of	60/043,576	04/11/97
PCT/US98/04482	Non-provisional of	60/047,501	05/23/97
PCT/US98/04482	Non-provisional of	60/043,670	04/11/97
PCT/US98/04482	Non-provisional of	60/056,632	08/22/97
PCT/US98/04482	Non-provisional of	60/056,664	08/22/97
PCT/US98/04482	Non-provisional of	60/056,876	08/22/97
PCT/US98/04482	Non-provisional of	60/056,881	08/22/97
PCT/US98/04482	Non-provisional of	60/056,909	08/22/97
PCT/US98/04482	Non-provisional of	60/056,875	08/22/97
PCT/US98/04482	Non-provisional of	60/056,862	08/22/97
PCT/US98/04482	Non-provisional of	60/056,887	08/22/97
PCT/US98/04482	Non-provisional of	60/056,908	08/22/97
PCT/US98/04482	Non-provisional of	60/048,964	06/06/97
PCT/US98/04482	Non-provisional of	60/057,650	09/05/97
PCT/US98/04482	Non-provisional of	60/056,884	08/22/97
10/100,683	Continuation-in-part of	09/882,171	06/18/01
09/882,171	Non-provisional of	60/190,068	03/17/00
09/882,171	Continuation of	09/809,391	03/16/01
09/809,391	Continuation-in-part of	09/149,476	09/08/98
09/149,476	Continuation-in-part of	PCT/US98/04493	03/06/98
10/100,683	Continuation-in-part of	09/809,391	03/16/01
09/809,391	Non-provisional of	60/190,068	03/17/00
09/809,391	Continuation-in-part of	09/149,476	09/08/98
09/149,476	Continuation-in-part of	PCT/US98/04493	03/06/98
10/100,683	Continuation-in-part of	09/149,476	09/08/98
09/149,476	Continuation-in-part of	PCT/US98/04493	03/06/98
10/100,683	Continuation-in-part of	PCT/US98/04493	03/06/98
PCT/US98/04493	Non-provisional of	60/040,161	03/07/97
PCT/US98/04493	Non-provisional of	60/040,162	03/07/97
PCT/US98/04493	Non-provisional of	60/040,333	03/07/97
PCT/US98/04493	Non-provisional of	60/038,621	03/07/97
PCT/US98/04493	Non-provisional of	60/040,626	03/07/97
PCT/US98/04493	Non-provisional of	60/040,334	03/07/97
PCT/US98/04493	Non-provisional of	60/040,336	03/07/97
PCT/US98/04493	Non-provisional of	60/040,163	03/07/97
PCT/US98/04493	Non-provisional of	60/047,600	05/23/97
PCT/US98/04493	Non-provisional of	60/047,615	05/23/97
PCT/US98/04493	Non-provisional of	60/047,597	05/23/97
PCT/US98/04493	Non-provisional of	60/047,502	05/23/97
PCT/US98/04493	Non-provisional of	60/047,633	05/23/97
PCT/US98/04493	Non-provisional of	60/047,583	05/23/97
PCT/US98/04493	Non-provisional of	60/047,617	05/23/97
PCT/US98/04493	Non-provisional of	60/047,618	05/23/97
PCT/US98/04493	Non-provisional of	60/047,503	05/23/97
PCT/US98/04493	Non-provisional of	60/047,592	05/23/97

PCT/US98/04493	Non-provisional of	60/047,581	05/23/97
PCT/US98/04493	Non-provisional of	60/047,584	05/23/97
PCT/US98/04493	Non-provisional of	60/047,500	05/23/97
PCT/US98/04493	Non-provisional of	60/047,587	05/23/97
PCT/US98/04493	Non-provisional of	60/047,492	05/23/97
PCT/US98/04493	Non-provisional of	60/047,598	05/23/97
PCT/US98/04493	Non-provisional of	60/047,613	05/23/97
PCT/US98/04493	Non-provisional of	60/047,582	05/23/97
PCT/US98/04493	Non-provisional of	60/047,596	05/23/97
PCT/US98/04493	Non-provisional of	60/047,612	05/23/97
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PCT/US98/04493	Non-provisional of	60/043,580	04/11/97
PCT/US98/04493	Non-provisional of	60/043,568	04/11/97
PCT/US98/04493	Non-provisional of	60/043,314	04/11/97
PCT/US98/04493	Non-provisional of	60/043,569	04/11/97
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PCT/US98/04493	Non-provisional of	60/043,674	04/11/97
PCT/US98/04493	Non-provisional of	60/043,669	04/11/97
PCT/US98/04493	Non-provisional of	60/043,312	04/11/97
PCT/US98/04493	Non-provisional of	60/043,313	04/11/97
PCT/US98/04493	Non-provisional of	60/043,672	04/11/97
PCT/US98/04493	Non-provisional of	60/043,315	04/11/97
PCT/US98/04493	Non-provisional of	60/048,974	06/06/97
PCT/US98/04493	Non-provisional of	60/056,886	08/22/97
PCT/US98/04493	Non-provisional of	60/056,877	08/22/97
PCT/US98/04493	Non-provisional of	60/056,889	08/22/97
PCT/US98/04493	Non-provisional of	60/056,893	08/22/97
PCT/US98/04493	Non-provisional of	60/056,630	08/22/97
PCT/US98/04493	Non-provisional of	60/056,878	08/22/97
PCT/US98/04493	Non-provisional of	60/056,662	08/22/97
PCT/US98/04493	Non-provisional of	60/056,872	08/22/97
PCT/US98/04493	Non-provisional of	60/056,882	08/22/97
PCT/US98/04493	Non-provisional of	60/056,637	08/22/97
PCT/US98/04493	Non-provisional of	60/056,903	08/22/97
PCT/US98/04493	Non-provisional of	60/056,888	08/22/97
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PCT/US98/04493	Non-provisional of	60/056,880	08/22/97
PCT/US98/04493	Non-provisional of	60/056,894	08/22/97
PCT/US98/04493	Non-provisional of	60/056,911	08/22/97
PCT/US98/04493	Non-provisional of	60/056,636	08/22/97
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PCT/US98/04493	Non-provisional of	60/056,910	08/22/97
PCT/US98/04493	Non-provisional of	60/056,864	08/22/97
PCT/US98/04493	Non-provisional of	60/056,631	08/22/97
PCT/US98/04493	Non-provisional of	60/056,845	08/22/97
PCT/US98/04493	Non-provisional of	60/056,892	08/22/97
PCT/US98/04493	Non-provisional of	60/057,761	09/05/97
PCT/US98/04493	Non-provisional of	60/047,595	05/23/97

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PCT/US98/04493	Non-provisional of	60/047,590	05/23/97
PCT/US98/04493	Non-provisional of	60/047,594	05/23/97
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PCT/US98/04493	Non-provisional of	60/047,593	05/23/97
PCT/US98/04493	Non-provisional of	60/047,614	05/23/97
PCT/US98/04493	Non-provisional of	60/043,578	04/11/97
PCT/US98/04493	Non-provisional of	60/043,576	04/11/97
PCT/US98/04493	Non-provisional of	60/047,501	05/23/97
PCT/US98/04493	Non-provisional of	60/043,670	04/11/97
PCT/US98/04493	Non-provisional of	60/056,632	08/22/97
PCT/US98/04493	Non-provisional of	60/056,664	08/22/97
PCT/US98/04493	Non-provisional of	60/056,876	08/22/97
PCT/US98/04493	Non-provisional of	60/056,881	08/22/97
PCT/US98/04493	Non-provisional of	60/056,909	08/22/97
PCT/US98/04493	Non-provisional of	60/056,875	08/22/97
PCT/US98/04493	Non-provisional of	60/056,862	08/22/97
PCT/US98/04493	Non-provisional of	60/056,887	08/22/97
PCT/US98/04493	Non-provisional of	60/056,908	08/22/97
PCT/US98/04493	Non-provisional of	60/048,964	06/06/97
PCT/US98/04493	Non-provisional of	60/057,650	09/05/97
PCT/US98/04493	Non-provisional of	60/056,884	08/22/97
PCT/US98/04493	Non-provisional of	60/057,669	09/05/97
PCT/US98/04493	Non-provisional of	60/049,610	06/13/97
PCT/US98/04493	Non-provisional of	60/061,060	10/02/97
PCT/US98/04493	Non-provisional of	60/051,926	07/08/97
PCT/US98/04493	Non-provisional of	60/052,874	07/16/97
PCT/US98/04493	Non-provisional of	60/058,785	09/12/97
PCT/US98/04493	Non-provisional of	60/055,724	08/18/97
10/100,683	Continuation-in-part of	10/058,993	01/30/02
10/058,993	Non-provisional of	60/265,583	02/02/01
10/058,993	Continuation-in-part of	09/852,659	05/11/01
09/852,659	Continuation-in-part of	09/152,060	09/11/98
09/152,060	Continuation-in-part of	PCT/US98/04858	03/12/98
10/058,993	Continuation-in-part of	09/853,161	05/11/01
09/853,161	Continuation-in-part of	09/152,060	09/11/98
09/152,060	Continuation-in-part of	PCT/US98/04858	03/12/98
10/058,993	Continuation-in-part of	09/852,797	05/11/01
09/852,797	Continuation-in-part of	09/152,060	09/11/98
09/152,060	Continuation-in-part of	PCT/US98/04858	03/12/98
10/100,683	Continuation-in-part of	09/852,659	05/11/01
09/852,659	Non-provisional of	60/265,583	02/02/01
09/852,659	Continuation-in-part of	09/152,060	09/11/98
09/152,060	Continuation-in-part of	PCT/US98/04858	03/12/98
10/100,683	Continuation-in-part of	09/853,161	05/11/01
09/853,161	Non-provisional of	60/265,583	02/02/01
09/853,161	Continuation-in-part of	09/152,060	09/11/98

09/152,060	Continuation-in-part of	PCT/US98/04858	03/12/98
10/100,683	Continuation-in-part of	09/852,797	05/11/01
09/852,797	Non-provisional of	60/265,583	02/02/01
09/852,797	Continuation-in-part of	09/152,060	09/11/98
09/152,060	Continuation-in-part of	PCT/US98/04858	03/12/98
10/100,683	Continuation-in-part of	09/152,060	09/11/98
09/152,060	Continuation-in-part of	PCT/US98/04858	03/12/98
10/100,683	Continuation-in-part of	PCT/US98/04858	03/12/98
PCT/US98/04858	Non-provisional of	60/040,762	03/14/97
PCT/US98/04858	Non-provisional of	60/040,710	03/14/97
PCT/US98/04858	Non-provisional of	60/050,934	05/30/97
PCT/US98/04858	Non-provisional of	60/048,100	05/30/97
PCT/US98/04858	Non-provisional of	60/048,357	05/30/97
PCT/US98/04858	Non-provisional of	60/048,189	05/30/97
PCT/US98/04858	Non-provisional of	60/057,765	09/05/97
PCT/US98/04858	Non-provisional of	60/048,970	06/06/97
PCT/US98/04858	Non-provisional of	60/068,368	12/19/97
10/100,683	Continuation-in-part of	10/059,395	01/31/02
10/059,395	Divisional of	09/966,262	10/01/01
09/966,262	Continuation of	09/154,707	09/17/98
09/154,707	Continuation-in-part of	PCT/US98/05311	03/19/98
10/100,683	Continuation-in-part of	09/984,245	10/29/01
09/984,245	Divisional of	09/154,707	09/17/98
09/154,707	Continuation-in-part of	PCT/US98/05311	03/19/98
10/100,683	Continuation-in-part of	09/983,966	10/26/01
09/983,966	Divisional of	09/154,707	09/17/98
09/154,707	Continuation-in-part of	PCT/US98/05311	03/19/98
10/100,683	Continuation-in-part of	09/966,262	10/01/01
09/966,262	Continuation of of	09/154,707	09/17/98
09/154,707	Continuation-in-part of	PCT/US98/05311	03/19/98
10/100,683	Continuation-in-part of	09/154,707	09/17/98
09/154,707	Continuation-in-part of	PCT/US98/05311	03/19/98
10/100,683	Continuation-in-part of	PCT/US98/05311	03/03/98
PCT/US98/05311	Non-provisional of	60/041,277	03/21/97
PCT/US98/05311	Non-provisional of	60/042,344	03/21/97
PCT/US98/05311	Non-provisional of	60/041,276	03/21/97
PCT/US98/05311	Non-provisional of	60/041,281	03/21/97
PCT/US98/05311	Non-provisional of	60/048,094	05/30/97
PCT/US98/05311	Non-provisional of	60/048,350	05/30/97
PCT/US98/05311	Non-provisional of	60/048,188	05/30/97
PCT/US98/05311	Non-provisional of	60/048,135	05/30/97
PCT/US98/05311	Non-provisional of	60/050,937	05/30/97
PCT/US98/05311	Non-provisional of	60/048,187	05/30/97
PCT/US98/05311	Non-provisional of	60/048,099	05/30/97
PCT/US98/05311	Non-provisional of	60/048,352	05/30/97
PCT/US98/05311	Non-provisional of	60/048,186	05/30/97
PCT/US98/05311	Non-provisional of	60/048,069	05/30/97
PCT/US98/05311	Non-provisional of	60/048,095	05/30/97
PCT/US98/05311	Non-provisional of	60/048,131	05/30/97
PCT/US98/05311	Non-provisional of	60/048,096	05/30/97

PCT/US98/05311	Non-provisional of	60/048,355	05/30/97
PCT/US98/05311	Non-provisional of	60/048,160	05/30/97
PCT/US98/05311	Non-provisional of	60/048,351	05/30/97
PCT/US98/05311	Non-provisional of	60/048,154	05/30/97
PCT/US98/05311	Non-provisional of	60/054,804	08/05/97
PCT/US98/05311	Non-provisional of	60/056,370	08/19/97
PCT/US98/05311	Non-provisional of	60/060,862	10/02/97
10/100,683	Continuation-in-part of	09/814,122	03/22/01
09/814,122	Continuation of	09/577,145	05/24/00
09/577,145	Continuation of	09/166,780	10/06/98
09/166,780	Continuation-in-part of	PCT/US98/06801	04/07/98
10/100,683	Continuation-in-part of	PCT/US98/06801	04/07/98
PCT/US98/06801	Non-provisional of	60/042,726	04/08/97
PCT/US98/06801	Non-provisional of	60/042,727	04/08/97
PCT/US98/06801	Non-provisional of	60/042,728	04/08/97
PCT/US98/06801	Non-provisional of	60/042,754	04/08/97
PCT/US98/06801	Non-provisional of	60/042,825	04/08/97
PCT/US98/06801	Non-provisional of	60/048,068	05/30/97
PCT/US98/06801	Non-provisional of	60/048,070	05/30/97
PCT/US98/06801	Non-provisional of	60/048,184	05/30/97
10/100,683	Continuation-in-part of	PCT/US98/06801	04/07/97
PCT/US98/06801	Non-provisional of	60/042,726	04/08/97
PCT/US98/06801	Non-provisional of	60/042,727	04/08/97
PCT/US98/06801	Non-provisional of	60/042,728	04/08/97
PCT/US98/06801	Non-provisional of	60/042,754	04/08/97
PCT/US98/06801	Non-provisional of	60/042,825	04/08/97
PCT/US98/06801	Non-provisional of	60/048,068	05/30/97
PCT/US98/06801	Non-provisional of	60/048,070	05/30/97
PCT/US98/06801	Non-provisional of	60/048,184	05/30/97
10/100,683	Continuation-in-part of	PCT/US98/10868	05/28/98
PCT/US98/10868	Non-provisional of	60/044,039	05/30/97
PCT/US98/10868	Non-provisional of	60/048,093	05/30/97
PCT/US98/10868	Non-provisional of	60/048,190	05/30/97
PCT/US98/10868	Non-provisional of	60/050,935	05/30/97
PCT/US98/10868	Non-provisional of	60/048,101	05/30/97
PCT/US98/10868	Non-provisional of	60/048,356	05/30/97
PCT/US98/10868	Non-provisional of	60/056,250	08/29/97
PCT/US98/10868	Non-provisional of	60/056,296	08/29/97
PCT/US98/10868	Non-provisional of	60/056,293	08/29/97
10/100,683	Continuation-in-part of	PCT/US98/11422	06/04/98
PCT/US98/11422	Non-provisional of	60/048,885	06/06/97
PCT/US98/11422	Non-provisional of	60/049,375	06/06/97
PCT/US98/11422	Non-provisional of	60/048,881	06/06/97
PCT/US98/11422	Non-provisional of	60/048,880	06/06/97
PCT/US98/11422	Non-provisional of	60/048,896	06/06/97
PCT/US98/11422	Non-provisional of	60/049,020	06/06/97
PCT/US98/11422	Non-provisional of	60/048,876	06/06/97
PCT/US98/11422	Non-provisional of	60/048,895	06/06/97
PCT/US98/11422	Non-provisional of	60/048,884	06/06/97
PCT/US98/11422	Non-provisional of	60/048,894	06/06/97

PCT/US98/11422	Non-provisional of	60/057,774	09/05/97
PCT/US98/11422	Non-provisional of	60/057,649	09/05/97
PCT/US98/11422	Non-provisional of	60/057,770	09/05/97
PCT/US98/11422	Non-provisional of	60/057,771	09/05/97
PCT/US98/11422	Non-provisional of	60/057,761	09/05/97
PCT/US98/11422	Non-provisional of	60/057,760	09/05/97
PCT/US98/11422	Non-provisional of	60/057,776	09/05/97
PCT/US98/11422	Non-provisional of	60/057,778	09/05/97
PCT/US98/11422	Non-provisional of	60/057,629	09/05/97
PCT/US98/11422	Non-provisional of	60/057,628	09/05/97
PCT/US98/11422	Non-provisional of	60/057,777	09/05/97
PCT/US98/11422	Non-provisional of	60/057,634	09/05/97
PCT/US98/11422	Non-provisional of	60/070,923	12/18/97
10/100,683	Continuation-in-part of	PCT/US01/05614	02/21/01
PCT/US01/05614	Non-provisional of	60/184,836	02/24/00
PCT/US01/05614	Non-provisional of	60/193,170	03/29/00
10/100,683	Continuation-in-part of	PCT/US98/12125	06/11/98
PCT/US98/12125	Non-provisional of	60/049,547	06/13/97
PCT/US98/12125	Non-provisional of	60/049,548	06/13/97
PCT/US98/12125	Non-provisional of	60/049,549	06/13/97
PCT/US98/12125	Non-provisional of	60/049,550	06/13/97
PCT/US98/12125	Non-provisional of	60/049,566	06/13/97
PCT/US98/12125	Non-provisional of	60/049,606	06/13/97
PCT/US98/12125	Non-provisional of	60/049,607	06/13/97
PCT/US98/12125	Non-provisional of	60/049,608	06/13/97
PCT/US98/12125	Non-provisional of	60/049,609	06/13/97
PCT/US98/12125	Non-provisional of	60/049,610	06/13/97
PCT/US98/12125	Non-provisional of	60/049,611	06/13/97
PCT/US98/12125	Non-provisional of	60/050,901	06/13/97
PCT/US98/12125	Non-provisional of	60/052,989	06/13/97
PCT/US98/12125	Non-provisional of	60/051,919	07/08/97
PCT/US98/12125	Non-provisional of	60/055,984	08/18/97
PCT/US98/12125	Non-provisional of	60/058,665	09/12/97
PCT/US98/12125	Non-provisional of	60/058,668	09/12/97
PCT/US98/12125	Non-provisional of	60/058,669	09/12/97
PCT/US98/12125	Non-provisional of	60/058,750	09/12/97
PCT/US98/12125	Non-provisional of	60/058,971	09/12/97
PCT/US98/12125	Non-provisional of	60/058,972	09/12/97
PCT/US98/12125	Non-provisional of	60/058,975	09/12/97
PCT/US98/12125	Non-provisional of	60/060,834	10/02/97
PCT/US98/12125	Non-provisional of	60/060,841	10/02/97
PCT/US98/12125	Non-provisional of	60/060,844	10/02/97
PCT/US98/12125	Non-provisional of	60/060,865	10/02/97
PCT/US98/12125	Non-provisional of	60/061,059	10/02/97
PCT/US98/12125	Non-provisional of	60/061,060	10/02/97
10/100,683	Continuation-in-part of	09/627,081	07/27/00
09/627,081	Continuation of	09/213,365	12/17/98
09/213,365	Continuation-in-part of	PCT/US98/13608	06/30/98
10/100,683	Continuation-in-part of	PCT/US98/13608	06/30/98
PCT/US98/13608	Non-provisional of	60/051,480	07/01/97

PCT/US98/13608	Non-provisional of	60/051,381	07/01/97
PCT/US98/13608	Non-provisional of	60/058,663	09/12/97
PCT/US98/13608	Non-provisional of	60/058,598	09/12/97
10/100,683	Continuation-in-part of	09/984,490	10/30/01
09/984,490	Divisional of	09/227,357	01/08/99
09/227,357	Continuation-in-part of	PCT/US98/13684	07/07/98
10/100,683	Continuation-in-part of	09/983,802	10/25/01
09/983,802	Continuation of	09/227,357	10/10/01
09/227,357	Continuation-in-part of	PCT/US98/13684	07/07/98
10/100,683	Continuation-in-part of	09/973,278	10/10/01
09/973,278	Non-provisional of	60/239,899	10/13/00
09/973,278	Continuation-in-part of	09/227,357	01/08/99
09/227,357	Continuation-in-part of	PCT/US98/13684	07/07/98
10/100,683	Continuation-in-part of	PCT/US98/13684	07/07/98
PCT/US98/13684	Non-provisional of	60/051,926	07/08/97
PCT/US98/13684	Non-provisional of	60/052,793	07/08/97
PCT/US98/13684	Non-provisional of	60/051,925	07/08/97
PCT/US98/13684	Non-provisional of	60/051,929	07/08/97
PCT/US98/13684	Non-provisional of	60/052,803	07/08/97
PCT/US98/13684	Non-provisional of	60/052,732	07/08/97
PCT/US98/13684	Non-provisional of	60/051,931	07/08/97
PCT/US98/13684	Non-provisional of	60/051,932	07/08/97
PCT/US98/13684	Non-provisional of	60/051,916	07/08/97
PCT/US98/13684	Non-provisional of	60/051,930	07/08/97
PCT/US98/13684	Non-provisional of	60/051,918	07/08/97
PCT/US98/13684	Non-provisional of	60/051,920	07/08/97
PCT/US98/13684	Non-provisional of	60/052,733	07/08/97
PCT/US98/13684	Non-provisional of	60/052,795	07/08/97
PCT/US98/13684	Non-provisional of	60/051,919	07/08/97
PCT/US98/13684	Non-provisional of	60/051,928	07/08/97
PCT/US98/13684	Non-provisional of	60/055,722	08/18/97
PCT/US98/13684	Non-provisional of	60/055,723	08/18/97
PCT/US98/13684	Non-provisional of	60/055,948	08/18/97
PCT/US98/13684	Non-provisional of	60/055,949	08/18/97
PCT/US98/13684	Non-provisional of	60/055,953	08/18/97
PCT/US98/13684	Non-provisional of	60/055,950	08/18/97
PCT/US98/13684	Non-provisional of	60/055,947	08/18/97
PCT/US98/13684	Non-provisional of	60/055,964	08/18/97
PCT/US98/13684	Non-provisional of	60/056,360	08/18/97
PCT/US98/13684	Non-provisional of	60/055,684	08/18/97
PCT/US98/13684	Non-provisional of	60/055,984	08/18/97
PCT/US98/13684	Non-provisional of	60/055,954	08/18/97
PCT/US98/13684	Non-provisional of	60/058,785	09/12/97
PCT/US98/13684	Non-provisional of	60/058,664	09/12/97
PCT/US98/13684	Non-provisional of	60/058,660	09/12/97
PCT/US98/13684	Non-provisional of	60/058,661	09/12/97
10/100,683	Continuation-in-part of	09/776,724	02/06/01
09/776,724	Non-provisional of	60/180,909	02/08/00
09/776,724	Continuation-in-part of	09/669,688	09/26/00
09/669,688	Continuation of	09/229,982	01/14/99

09/229,982	Continuation-in-part of	PCT/US98/14613	07/15/98
10/100,683	Continuation-in-part of	09/669,688	09/26/00
09/669,688	Continuation of	09/229,982	01/14/99
09/229,982	Continuation-in-part of	PCT/US98/14613	07/15/98
10/100,683	Continuation-in-part of	09/229,982	01/14/99
09/229,982	Continuation-in-part of	PCT/US98/14613	07/15/98
10/100,683	Continuation-in-part of	PCT/US98/14613	07/15/98
PCT/US98/14613	Non-provisional of	60/052,661	07/16/97
PCT/US98/14613	Non-provisional of	60/052,872	07/16/97
PCT/US98/14613	Non-provisional of	60/052,871	07/16/97
PCT/US98/14613	Non-provisional of	60/052,874	07/16/97
PCT/US98/14613	Non-provisional of	60/052,873	07/16/97
PCT/US98/14613	Non-provisional of	60/052,870	07/16/97
PCT/US98/14613	Non-provisional of	60/052,875	07/16/97
PCT/US98/14613	Non-provisional of	60/053,440	07/22/97
PCT/US98/14613	Non-provisional of	60/053,441	07/22/97
PCT/US98/14613	Non-provisional of	60/053,442	07/22/97
PCT/US98/14613	Non-provisional of	60/056,359	08/18/97
PCT/US98/14613	Non-provisional of	60/055,725	08/18/97
PCT/US98/14613	Non-provisional of	60/055,985	08/18/97
PCT/US98/14613	Non-provisional of	60/055,952	08/18/97
PCT/US98/14613	Non-provisional of	60/055,989	08/18/97
PCT/US98/14613	Non-provisional of	60/056,361	08/18/97
PCT/US98/14613	Non-provisional of	60/055,726	08/18/97
PCT/US98/14613	Non-provisional of	60/055,724	08/18/97
PCT/US98/14613	Non-provisional of	60/055,946	08/18/97
PCT/US98/14613	Non-provisional of	60/055,683	08/18/97
10/100,683	Non-provisional of	60/295,558	06/05/01
10/100,683	Continuation-in-part of	09/820,649	03/30/01
09/820,649	Continuation of	09/666,984	09/21/00
09/666,984	Continuation of	09/236,557	01/26/99
09/236,557	Continuation-in-part of	PCT/US98/15949	07/29/98
10/100,683	Continuation-in-part of	PCT/US98/15949	07/29/98
PCT/US98/15949	Non-provisional of	60/054,212	07/30/97
PCT/US98/15949	Non-provisional of	60/054,209	07/30/97
PCT/US98/15949	Non-provisional of	60/054,234	07/30/97
PCT/US98/15949	Non-provisional of	60/054,218	07/30/97
PCT/US98/15949	Non-provisional of	60/054,214	07/30/97
PCT/US98/15949	Non-provisional of	60/054,236	07/30/97
PCT/US98/15949	Non-provisional of	60/054,215	07/30/97
PCT/US98/15949	Non-provisional of	60/054,211	07/30/97
PCT/US98/15949	Non-provisional of	60/054,217	07/30/97
PCT/US98/15949	Non-provisional of	60/054,213	07/30/97
PCT/US98/15949	Non-provisional of	60/055,968	08/18/97
PCT/US98/15949	Non-provisional of	60/055,969	08/18/97
PCT/US98/15949	Non-provisional of	60/055,972	08/18/97
PCT/US98/15949	Non-provisional of	60/056,561	08/19/97
PCT/US98/15949	Non-provisional of	60/056,534	08/19/97
PCT/US98/15949	Non-provisional of	60/056,729	08/19/97
PCT/US98/15949	Non-provisional of	60/056,543	08/19/97

PCT/US98/15949	Non-provisional of	60/056,727	08/19/97
PCT/US98/15949	Non-provisional of	60/056,554	08/19/97
PCT/US98/15949	Non-provisional of	60/056,730	08/19/97
10/100,683	Continuation-in-part of	09/969,730	10/04/01
09/969,730	Continuation-in-part of	09/774,639	02/01/01
09/774,639	Continuation of	09/244,112	02/04/99
09/244,112	Continuation-in-part of	PCT/US98/16235	08/04/98
10/100,683	Continuation-in-part of	09/774,639	02/01/01
09/774,639	Continuation of	09/244,112	02/04/99
09/244,112	Continuation-in-part of	PCT/US98/16235	08/04/98
10/100,683	Continuation-in-part of	09/969,730	10/04/01
09/969,730	Non-provisional of	60/238,291	10/06/00
10/100,683	Continuation-in-part of	PCT/US98/16235	08/04/98
PCT/US98/16235	Non-provisional of	60/055,386	08/05/97
PCT/US98/16235	Non-provisional of	60/054,807	08/05/97
PCT/US98/16235	Non-provisional of	60/055,312	08/05/97
PCT/US98/16235	Non-provisional of	60/055,309	08/05/97
PCT/US98/16235	Non-provisional of	60/054,798	08/05/97
PCT/US98/16235	Non-provisional of	60/055,310	08/05/97
PCT/US98/16235	Non-provisional of	60/054,806	08/05/97
PCT/US98/16235	Non-provisional of	60/054,809	08/05/97
PCT/US98/16235	Non-provisional of	60/054,804	08/05/97
PCT/US98/16235	Non-provisional of	60/054,803	08/05/97
PCT/US98/16235	Non-provisional of	60/054,808	08/05/97
PCT/US98/16235	Non-provisional of	60/055,311	08/05/97
PCT/US98/16235	Non-provisional of	60/055,986	08/18/97
PCT/US98/16235	Non-provisional of	60/055,970	08/18/97
PCT/US98/16235	Non-provisional of	60/056,563	08/19/97
PCT/US98/16235	Non-provisional of	60/056,557	08/19/97
PCT/US98/16235	Non-provisional of	60/056,731	08/19/97
PCT/US98/16235	Non-provisional of	60/056,365	08/19/97
PCT/US98/16235	Non-provisional of	60/056,367	08/19/97
PCT/US98/16235	Non-provisional of	60/056,370	08/19/97
PCT/US98/16235	Non-provisional of	60/056,364	08/19/97
PCT/US98/16235	Non-provisional of	60/056,366	08/19/97
PCT/US98/16235	Non-provisional of	60/056,732	08/19/97
PCT/US98/16235	Non-provisional of	60/056,371	08/19/97
10/100,683	Continuation-in-part of	09/716,128	11/17/00
09/716,128	Continuation of	09/251,329	02/17/99
09/251,329	Continuation-in-part of	PCT/US98/17044	08/18/98
10/100,683	Continuation-in-part of	PCT/US98/17044	08/18/98
PCT/US98/17044	Non-provisional of	60/056,555	08/19/97
PCT/US98/17044	Non-provisional of	60/056,556	08/19/97
PCT/US98/17044	Non-provisional of	60/056,535	08/19/97
PCT/US98/17044	Non-provisional of	60/056,629	08/19/97
PCT/US98/17044	Non-provisional of	60/056,369	08/19/97
PCT/US98/17044	Non-provisional of	60/056,628	08/19/97
PCT/US98/17044	Non-provisional of	60/056,728	08/19/97
PCT/US98/17044	Non-provisional of	60/056,368	08/19/97
PCT/US98/17044	Non-provisional of	60/056,726	08/19/97

PCT/US98/17044	Non-provisional of	60/089,510	06/16/98
PCT/US98/17044	Non-provisional of	60/092,956	07/15/98
<u>10/100,683</u>	<u>Continuation-in-part of</u>	<u>09/729,835</u>	12/06/00
09/729,835	Divisional of	09/257,179	02/25/99
09/257,179	Continuation-in-part of	PCT/US98/17709	08/27/98
<u>10/100,683</u>	<u>Continuation-in-part of</u>	<u>09/257,179</u>	02/25/99
09/257,179	Continuation-in-part of	PCT/US98/17709	08/27/98
<u>10/100,683</u>	<u>Continuation-in-part of</u>	<u>PCT/US98/17709</u>	08/27/98
PCT/US98/17709	Non-provisional of	60/056,270	08/29/97
PCT/US98/17709	Non-provisional of	60/056,271	08/29/97
PCT/US98/17709	Non-provisional of	60/056,247	08/29/97
PCT/US98/17709	Non-provisional of	60/056,073	08/29/97
10/100,683	Continuation-in-part of	10/047,021	01/17/02
10/047,021	Continuation-in-part of	09/722,329	11/28/00
09/722,329	Continuation of	09/262,109	03/04/99
09/262,109	Continuation-in-part of	PCT/US98/18360	09/03/98
10/100,683	Continuation-in-part of	09/722,329	11/28/00
09/722,329	Continuation of	09/262,109	03/04/99
09/262,109	Continuation-in-part of	PCT/US98/18360	09/03/98
10/100,683	Continuation-in-part of	PZ016pct2	01/17/02
PZ016pct2	Non-provisional of	60/262,066	01/18/01
10/100,683	Continuation-in-part of	PCT/US98/18360	09/03/98
PCT/US98/18360	Non-provisional of	60/057,626	09/05/97
PCT/US98/18360	Non-provisional of	60/057,663	09/05/97
PCT/US98/18360	Non-provisional of	60/057,669	09/05/97
PCT/US98/18360	Non-provisional of	60/058,667	09/12/97
PCT/US98/18360	Non-provisional of	60/058,974	09/12/97
PCT/US98/18360	Non-provisional of	60/058,973	09/12/97
PCT/US98/18360	Non-provisional of	60/058,666	09/12/97
PCT/US98/18360	Non-provisional of	60/090,112	06/22/98
10/100,683	Continuation-in-part of	09/281,976	03/31/99
09/281,976	Continuation-in-part of	PCT/US98/20775	10/01/98
10/100,683	Continuation-in-part of	PCT/US98/20775	10/01/98
PCT/US98/20775	Non-provisional of	60/060,837	10/02/97
PCT/US98/20775	Non-provisional of	60/060,862	10/02/97
PCT/US98/20775	Non-provisional of	60/060,839	10/02/97
PCT/US98/20775	Non-provisional of	60/060,866	10/02/97
PCT/US98/20775	Non-provisional of	60/060,843	10/02/97
PCT/US98/20775	Non-provisional of	60/060,836	10/02/97
PCT/US98/20775	Non-provisional of	60/060,838	10/02/97
PCT/US98/20775	Non-provisional of	60/060,874	10/02/97
PCT/US98/20775	Non-provisional of	60/060,833	10/02/97
PCT/US98/20775	Non-provisional of	60/060,884	10/02/97
PCT/US98/20775	Non-provisional of	60/060,880	10/02/97
<u>10/100,683</u>	<u>Continuation-in-part of</u>	<u>09/984,429</u>	10/30/01
09/984,429	Non-provisional of	60/244,591	11/01/00
09/984,429	Continuation-in-part of	09/288,143	04/08/99

09/288,143	Continuation-in-part of	PCT/US98/21142	10/08/98
10/100,683	Non-provisional of	60/244,591	11/01/00
10/100,683	Continuation-in-part of	09/288,143	04/08/99
09/288,143	Continuation-in-part of	PCT/US98/21142	10/08/98
10/100,683	Continuation-in-part of	PCT/US98/21142	10/08/98
PCT/US98/21142	Non-provisional of	60/061,463	10/09/97
PCT/US98/21142	Non-provisional of	60/061,529	10/09/97
PCT/US98/21142	Non-provisional of	60/071,498	10/09/97
PCT/US98/21142	Non-provisional of	60/061,527	10/09/97
PCT/US98/21142	Non-provisional of	60/061,536	10/09/97
PCT/US98/21142	Non-provisional of	60/061,532	10/09/97
10/100,683	Continuation-in-part of	09/296,622	04/23/99
09/296,622	Continuation-in-part of	PCT/US98/22376	10/23/98
10/100,683	Continuation-in-part of	PCT/US98/22376	10/23/98
PCT/US98/22376	Non-provisional of	60/063,099	10/24/97
PCT/US98/22376	Non-provisional of	60/063,088	10/24/97
PCT/US98/22376	Non-provisional of	60/063,100	10/24/97
PCT/US98/22376	Non-provisional of	60/063,387	10/24/97
PCT/US98/22376	Non-provisional of	60/063,148	10/24/97
PCT/US98/22376	Non-provisional of	60/063,386	10/24/97
PCT/US98/22376	Non-provisional of	60/062,784	10/24/97
PCT/US98/22376	Non-provisional of	60/063,091	10/24/97
PCT/US98/22376	Non-provisional of	60/063,090	10/24/97
PCT/US98/22376	Non-provisional of	60/063,089	10/24/97
PCT/US98/22376	Non-provisional of	60/063,092	10/24/97
PCT/US98/22376	Non-provisional of	60/063,111	10/24/97
PCT/US98/22376	Non-provisional of	60/063,101	10/24/97
PCT/US98/22376	Non-provisional of	60/063,109	10/24/97
PCT/US98/22376	Non-provisional of	60/063,110	10/24/97
PCT/US98/22376	Non-provisional of	60/063,098	10/24/97
PCT/US98/22376	Non-provisional of	60/063,097	10/24/97
10/100,683	Continuation-in-part of	09/974,879	10/12/01
09/974,879	Non-provisional of	60/239,893	10/13/00
09/974,879	Continuation-in-part of	09/818,683	03/28/01
09/818,683	Continuation of	09/305,736	05/05/99
09/305,736	Continuation-in-part of	PCT/US98/23435	11/04/98
10/100,683	Continuation-in-part of	09/818,683	03/28/01
09/818,683	Continuation of	09/305,736	05/05/99
09/305,736	Continuation-in-part of	PCT/US98/23435	11/04/98
10/100,683	Continuation-in-part of	09/305,736	05/05/99
09/305,736	Continuation-in-part of	PCT/US98/23435	11/04/98
10/100,683	Continuation-in-part of	PCT/US98/23435	11/04/98
PCT/US98/23435	Non-provisional of	60/064,911	11/07/97
PCT/US98/23435	Non-provisional of	60/064,912	11/07/97
PCT/US98/23435	Non-provisional of	60/064,983	11/07/97
PCT/US98/23435	Non-provisional of	60/064,900	11/07/97
PCT/US98/23435	Non-provisional of	60/064,988	11/07/97
PCT/US98/23435	Non-provisional of	60/064,987	11/07/97

PCT/US98/23435	Non-provisional of	60/064,908	11/07/97
PCT/US98/23435	Non-provisional of	60/064,984	11/07/97
PCT/US98/23435	Non-provisional of	60/064,985	11/07/97
PCT/US98/23435	Non-provisional of	60/066,094	11/17/97
PCT/US98/23435	Non-provisional of	60/066,100	11/17/97
PCT/US98/23435	Non-provisional of	60/066,089	11/17/97
PCT/US98/23435	Non-provisional of	60/066,095	11/17/97
PCT/US98/23435	Non-provisional of	60/066,090	11/17/97
10/100,683	Continuation-in-part of	09/334,595	06/17/99
09/334,595	Continuation-in-part of	PCT/US98/27059	12/17/98
10/100,683	Continuation-in-part of	PCT/US98/27059	12/17/98
PCT/US98/27059	Non-provisional of	60/070,923	12/18/97
PCT/US98/27059	Non-provisional of	60/068,007	12/18/97
PCT/US98/27059	Non-provisional of	60/068,057	12/18/97
PCT/US98/27059	Non-provisional of	60/068,006	12/18/97
PCT/US98/27059	Non-provisional of	60/068,369	12/19/97
PCT/US98/27059	Non-provisional of	60/068,367	12/19/97
PCT/US98/27059	Non-provisional of	60/068,368	12/19/97
PCT/US98/27059	Non-provisional of	60/068,169	12/19/97
PCT/US98/27059	Non-provisional of	60/068,053	12/18/97
PCT/US98/27059	Non-provisional of	60/068,064	12/18/97
PCT/US98/27059	Non-provisional of	60/068,054	12/18/97
PCT/US98/27059	Non-provisional of	60/068,008	12/18/97
PCT/US98/27059	Non-provisional of	60/068,365	12/19/97
10/100,683	Continuation-in-part of	09/938,671	08/27/01
09/938,671	Continuation of	09/739,907	12/20/00
09/739,907	Continuation of	09/348,457	07/07/99
09/348,457	Continuation-in-part of	PCT/US99/00108	01/06/99
10/100,683	Continuation-in-part of	09/739,907	12/20/00
09/739,907	Continuation of	09/348,457	07/07/99
09/348,457	Continuation-in-part of	PCT/US99/00108	01/06/99
10/100,683	Continuation-in-part of	09/348,457	07/07/99
09/348,457	Continuation-in-part of	PCT/US99/00108	01/06/99
10/100,683	Continuation-in-part of	PCT/US99/00108	01/06/99
PCT/US99/00108	Non-provisional of	60/070,704	01/07/98
PCT/US99/00108	Non-provisional of	60/070,658	01/07/98
PCT/US99/00108	Non-provisional of	60/070,692	01/07/98
PCT/US99/00108	Non-provisional of	60/070,657	01/07/98
10/100,683	Continuation-in-part of	09/949,925	09/12/01
09/949,925	Non-provisional of	60/232,150	09/12/00
09/949,925	Continuation-in-part of	PCT/US99/01621	01/27/99
09/949,925	Continuation-in-part of	09/363,044	07/29/99
09/363,044	Continuation-in-part of	PCT/US99/01621	01/27/99
10/100,683	Continuation-in-part of	09/813,153	03/21/01
09/813,153	Continuation of	09/363,044	07/29/99
09/363,044	Continuation-in-part of	PCT/US99/01621	01/27/99

10/100,683	Continuation-in-part of	09/363,044	07/29/99
09/363,044	Continuation-in-part of	PCT/US99/01621	01/27/99
10/100,683	Continuation-in-part of	PCT/US99/01621	01/27/99
PCT/US99/01621	Non-provisional of	60/073,170	01/30/98
PCT/US99/01621	Non-provisional of	60/073,167	01/30/98
PCT/US99/01621	Non-provisional of	60/073,165	01/30/98
PCT/US99/01621	Non-provisional of	60/073,164	01/30/98
PCT/US99/01621	Non-provisional of	60/073,162	01/30/98
PCT/US99/01621	Non-provisional of	60/073,161	01/30/98
PCT/US99/01621	Non-provisional of	60/073,160	01/30/98
PCT/US99/01621	Non-provisional of	60/073,159	01/30/98
10/100,683	Continuation-in-part of	10/062,548	02/05/02
10/062,548	Continuation of	09/369,247	08/05/99
09/369,247	Continuation-in-part of	PCT/US99/02293	02/04/99
10/100,683	Continuation-in-part of	09/369,247	08/05/99
09/369,247	Continuation-in-part of	PCT/US99/02293	02/04/99
10/100,683	Continuation-in-part of	PCT/US99/02293	02/04/99
PCT/US99/02293	Non-provisional of	60/074,118	02/09/98
PCT/US99/02293	Non-provisional of	60/074,157	02/09/98
PCT/US99/02293	Non-provisional of	60/074,037	02/09/98
PCT/US99/02293	Non-provisional of	60/074,141	02/09/98
PCT/US99/02293	Non-provisional of	60/074,341	02/09/98
10/100,683	Continuation-in-part of	09/716,129	11/17/00
09/716,129	Continuation-in-part of	PCT/US99/03939	02/24/99
09/716,129	CON	09/382,572	08/25/99
09/382,572	Continuation-in-part of	PCT/US99/03939	02/24/99
10/100,683	Continuation-in-part of	PCT/US99/03939	02/24/99
PCT/US99/03939	Non-provisional of	60/076,053	02/26/98
PCT/US99/03939	Non-provisional of	60/076,051	02/26/98
PCT/US99/03939	Non-provisional of	60/076,054	02/26/98
PCT/US99/03939	Non-provisional of	60/076,052	02/26/98
PCT/US99/03939	Non-provisional of	60/076,057	02/26/98
10/100,683	Continuation-in-part of	09/798,889	03/06/01
09/798,889	CON	09/393,022	09/09/99
09/393,022	Continuation-in-part of	PCT/US99/05721	03/11/99
10/100,683	Continuation-in-part of	PCT/US99/05721	03/11/99
PCT/US99/05721	Non-provisional of	60/077,714	03/12/98
PCT/US99/05721	Non-provisional of	60/077,686	03/12/98
PCT/US99/05721	Non-provisional of	60/077,687	03/12/98
PCT/US99/05721	Non-provisional of	60/077,696	03/12/98
10/100,683	Continuation-in-part of	09/397,945	09/17/99
09/397,945	Continuation-in-part of	PCT/US99/05804	03/18/99
10/100,683	Continuation-in-part of	PCT/US99/05804	03/18/99
PCT/US99/05804	Non-provisional of	60/078,566	03/19/98
PCT/US99/05804	Non-provisional of	60/078,576	03/19/98
PCT/US99/05804	Non-provisional of	60/078,573	03/19/98
PCT/US99/05804	Non-provisional of	60/078,574	03/19/98
PCT/US99/05804	Non-provisional of	60/078,579	03/19/98

PCT/US99/05804	Non-provisional of	60/080,314	04/01/98
PCT/US99/05804	Non-provisional of	60/080,312	04/01/98
PCT/US99/05804	Non-provisional of	60/078,578	03/19/98
PCT/US99/05804	Non-provisional of	60/078,581	03/19/98
PCT/US99/05804	Non-provisional of	60/078,577	03/19/98
PCT/US99/05804	Non-provisional of	60/078,563	03/19/98
PCT/US99/05804	Non-provisional of	60/080,313	04/01/98
10/100,683	Continuation-in-part of	09/948,783	09/10/01
<u>09/948,783</u>	Non-provisional of	<u>60/231,846</u>	09/11/00
<u>09/948,783</u>	Continuation-in-part of	<u>09/892,877</u>	06/28/01
<u>09/892,877</u>	Continuation of	<u>09/437,658</u>	11/10/99
09/437,658	Continuation-in-part of	PCT/US99/09847	05/06/99
<u>10/100,683</u>	Continuation-in-part of	<u>09/892,877</u>	06/28/01
<u>09/892,877</u>	Continuation of	<u>09/437,658</u>	11/10/99
09/437,658	Continuation-in-part of	PCT/US99/09847	05/06/99
<u>10/100,683</u>	Continuation-in-part of	<u>PCT/US99/09847</u>	05/06/99
PCT/US99/09847	Non-provisional of	60/085,093	05/12/98
PCT/US99/09847	Non-provisional of	60/085,094	05/12/98
PCT/US99/09847	Non-provisional of	60/085,105	05/12/98
PCT/US99/09847	Non-provisional of	60/085,180	05/12/98
PCT/US99/09847	Non-provisional of	60/085,927	05/18/98
PCT/US99/09847	Non-provisional of	60/085,906	05/18/98
PCT/US99/09847	Non-provisional of	60/085,920	05/18/98
PCT/US99/09847	Non-provisional of	60/085,924	05/18/98
PCT/US99/09847	Non-provisional of	60/085,922	05/18/98
PCT/US99/09847	Non-provisional of	60/085,923	05/18/98
PCT/US99/09847	Non-provisional of	60/085,921	05/18/98
PCT/US99/09847	Non-provisional of	60/085,925	05/18/98
PCT/US99/09847	Non-provisional of	60/085,928	05/18/98
10/100,683	Continuation-in-part of	10/050,873	01/18/02
10/050,873	Non-provisional of	60/263,681	01/24/01
10/050,873	Non-provisional of	60/263,230	01/23/01
10/050,873	Continuation-in-part of	09/461,325	12/14/99
09/461,325	Continuation-in-part of	PCT/US99/13418	06/15/99
10/100,683	Continuation-in-part of	10/012,542	12/12/01
10/012,542	Divisional of	09/461,325	12/14/99
09/461,325	Continuation-in-part of	PCT/US99/13418	06/15/99
<u>10/100,683</u>	Continuation-in-part of	<u>09/461,325</u>	12/14/99
09/461,325	Continuation-in-part of	PCT/US99/13418	06/15/99
10/100,683	Continuation-in-part of	PCT/US99/13418	06/15/99
PCT/US99/13418	Non-provisional of	60/089,507	06/16/98
PCT/US99/13418	Non-provisional of	60/089,508	06/16/98
PCT/US99/13418	Non-provisional of	60/089,509	06/16/98
PCT/US99/13418	Non-provisional of	60/089,510	06/16/98
PCT/US99/13418	Non-provisional of	60/090,112	06/22/98

PCT/US99/13418	Non-provisional of	60/090,113	06/22/98
10/100,683	Continuation-in-part of	09/984,271	10/29/01
09/984,271	Divisional of	09/482,273	01/13/00
09/482,273	Continuation-in-part of	PCT/US99/15849	07/14/99
10/100,683	Continuation-in-part of	09/984,276	10/29/01
09/984,276	Divisional of	09/482,273	01/13/00
09/482,273	Continuation-in-part of	PCT/US99/15849	07/14/99
10/100,683	Continuation-in-part of	09/482,273	01/13/00
09/482,273	Continuation-in-part of	PCT/US99/15849	07/14/99
10/100,683	Continuation-in-part of	PCT/US99/15849	07/14/99
PCT/US99/15849	Non-provisional of	60/092,921	07/15/98
PCT/US99/15849	Non-provisional of	60/092,922	07/15/98
PCT/US99/15849	Non-provisional of	60/092,956	07/15/98
10/100,683	Continuation-in-part of	PCT/US01/29871	09/24/01
PCT/US01/29871	Non-provisional of	60/234,925	09/25/00
PCT/US01/29871	Continuation-in-part of	PCT/US01/00911	01/12/01
10/100,683	Continuation-in-part of	PCT/US01/00911	01/12/01
PCT/US01/00911	Continuation-in-part of	09/482,273	01/13/00
10/100,683	Non-provisional of	60/350,898	01/25/02
10/100,683	Continuation-in-part of	09/489,847	01/24/00
09/489,847	Continuation-in-part of	PCT/US99/17130	07/29/99
10/100,683	Continuation-in-part of	PCT/US99/17130	07/29/99
PCT/US99/17130	Non-provisional of	60/094,657	07/30/98
PCT/US99/17130	Non-provisional of	60/095,486	08/05/98
PCT/US99/17130	Non-provisional of	60/096,319	08/12/98
PCT/US99/17130	Non-provisional of	60/095,454	08/06/98
PCT/US99/17130	Non-provisional of	60/095,455	08/06/98
10/100,683	Continuation-in-part of	10/054,988	01/25/02
10/054,988	Continuation of	09/904,615	07/16/01
09/904,615	Continuation of	09/739,254	12/19/00
09/739,254	Continuation of	09/511,554	02/23/00
09/511,554	Continuation-in-part of	PCT/US99/19330	08/24/99
10/100,683	Continuation-in-part of	09/904,615	07/16/01
09/904,615	Continuation of	09/739,254	12/19/00
09/739,254	Continuation of	09/511,554	02/23/00
09/511,554	Continuation-in-part of	PCT/US99/19330	08/24/99
10/100,683	Continuation-in-part of	PCT/US99/19330	08/24/99
PCT/US99/19330	Non-provisional of	60/097,917	08/25/98
PCT/US99/19330	Non-provisional of	60/098,634	08/31/98
10/100,683	Continuation-in-part of	09/820,893	03/30/01
09/820,893	Continuation of	09/531,119	03/20/00
09/531,119	Continuation-in-part of	PCT/US99/22012	09/22/99
10/100,683	Continuation-in-part of	PCT/US99/22012	09/22/99
PCT/US99/22012	Non-provisional of	60/101,546	09/23/98
PCT/US99/22012	Non-provisional of	60/102,895	10/02/98
10/100,683	Continuation-in-part of	09/948,820	09/10/01
09/948,820	Continuation of	09/565,391	05/05/00
09/565,391	Continuation-in-part of	PCT/US99/26409	11/09/99
10/100,683	Continuation-in-part of	09/565,391	05/05/00
09/565,391	Continuation-in-part of	PCT/US99/26409	11/09/99

10/100,683	Continuation-in-part of	PCT/US99/26409	11/09/99
PCT/US99/26409	Non-provisional of	60/108,207	11/12/98
10/100,683	Continuation-in-part of	09/895,298	07/02/01
09/895,298	Continuation of	09/591,316	06/09/00
09/591,316	Continuation-in-part of	PCT/US99/29950	12/16/99
10/100,683	Continuation-in-part of	PCT/US99/29950	12/16/99
PCT/US99/29950	Non-provisional of	60/113,006	12/18/98
PCT/US99/29950	Non-provisional of	60/112,809	12/17/98
10/100,683	Continuation-in-part of	09/985,153	11/01/01
09/985,153	Continuation of	09/618,150	07/17/00
09/618,150	Continuation-in-part of	PCT/US00/00903	01/18/00
10/100,683	Continuation-in-part of	PCT/US00/00903	01/18/00
PCT/US00/00903	Non-provisional of	60/116,330	01/19/99
10/100,683	Continuation-in-part of	09/997,131	11/30/01
09/997,131	Continuation of	09/628,508	07/28/00
09/628,508	Continuation-in-part of	PCT/US00/03062	02/08/00
10/100,683	Continuation-in-part of	PCT/US00/03062	02/08/00
PCT/US00/03062	Non-provisional of	60/119,468	02/10/99
10/100,683	Continuation-in-part of	10/050,882	01/18/02
10/050,882	Continuation of	09/661,453	09/13/00
09/661,453	Continuation-in-part of	PCT/US00/06783	03/16/00
10/100,683	Continuation-in-part of	09/661,453	09/13/00
09/661,453	Continuation-in-part of	PCT/US00/06783	03/16/00
10/100,683	Continuation-in-part of	PCT/US00/06783	03/16/00
PCT/US00/06783	Non-provisional of	60/125,055	03/18/99
10/100,683	Continuation-in-part of	10/050,704	01/18/02
10/050,704	Continuation of	09/684,524	10/10/00
09/684,524	Continuation-in-part of	PCT/US00/08979	04/06/00
10/100,683	Continuation-in-part of	09/684,524	10/10/00
09/684,524	Continuation-in-part of	PCT/US00/08979	04/06/00
10/100,683	Continuation-in-part of	PCT/US00/08979	04/06/00
PCT/US00/08979	Non-provisional of	60/128,693	04/09/99
PCT/US00/08979	Non-provisional of	60/130,991	04/26/99
10/100,683	Continuation-in-part of	10/042,141	01/11/02
10/042,141	Continuation of	09/726,643	12/01/00
09/726,643	Continuation-in-part of	PCT/US00/15187	06/02/00
10/100,683	Continuation-in-part of	09/726,643	12/01/00
09/726,643	Continuation-in-part of	PCT/US00/15187	06/02/00
10/100,683	Continuation-in-part of	PCT/US00/15187	06/02/00
PCT/US00/15187	Non-provisional of	60/137,725	06/07/99
10/100,683	Continuation-in-part of	09/756,168	01/09/01
09/756,168	Continuation-in-part of	PCT/US00/19735	07/23/99
10/100,683	Continuation-in-part of	PCT/US00/19735	07/20/00
PCT/US00/19735	Non-provisional of	60/145,220	07/23/99
10/100,683	Continuation-in-part of	PZ042P1C1	02/01/02
PZ042P1C1	Continuation of	09/781,417	02/13/01
09/781,417	Continuation-in-part of	PCT/US00/22325	08/16/00
10/100,683	Continuation-in-part of	09/781,417	02/13/01
09/781,417	Continuation-in-part of	PCT/US00/22325	08/16/00

10/100,683	Continuation-in-part of	PCT/US00/22325	08/16/00
PCT/US00/22325	Non-provisional of	60/149,182	08/17/99
10/100,683	Continuation-in-part of	09/789,561	02/22/01
09/789,561	Continuation-in-part of	PCT/US00/24008	08/31/00
10/100,683	Continuation-in-part of	PCT/US00/24008	08/31/00
PCT/US00/24008	Non-provisional of	60/152,315	09/03/99
PCT/US00/24008	Non-provisional of	60/152,317	09/03/99
10/100,683	Continuation-in-part of	09/800,729	03/08/01
09/800,729	Continuation-in-part of	PCT/US00/26013	09/22/00
10/100,683	Continuation-in-part of	PCT/US00/26013	09/22/00
PCT/US00/26013	Non-provisional of	60/155,709	09/24/99
10/100,683	Continuation-in-part of	09/832,129	04/11/01
09/832,129	Continuation-in-part of	PCT/US00/28664	10/17/00
10/100,683	Continuation-in-part of	PCT/US00/28664	10/17/00
PCT/US00/28664	Non-provisional of	60/163,085	11/02/99
PCT/US00/28664	Non-provisional of	60/172,411	12/17/99
10/100,683	Continuation-in-part of	PCT/US00/29363	10/25/00
PCT/US00/29363	Non-provisional of	60/215,139	06/30/00
PCT/US00/29363	Non-provisional of	60/162,239	10/29/99
10/100,683	Continuation-in-part of	PCT/US00/29360	10/25/00
PCT/US00/29360	Non-provisional of	60/215,138	06/30/00
PCT/US00/29360	Non-provisional of	60/162,211	10/29/99
10/100,683	Continuation-in-part of	PCT/US00/29362	10/25/00
PCT/US00/29362	Non-provisional of	60/215,131	06/30/00
PCT/US00/29362	Non-provisional of	60/162,240	10/29/99
10/100,683	Continuation-in-part of	PCT/US00/29365	10/25/00
PCT/US00/29365	Non-provisional of	60/219,666	07/21/00
PCT/US00/29365	Non-provisional of	60/162,237	10/29/99
10/100,683	Continuation-in-part of	PCT/US00/29364	10/25/00
PCT/US00/29364	Non-provisional of	60/215,134	06/30/00
PCT/US00/29364	Non-provisional of	60/162,238	10/29/99
10/100,683	Continuation-in-part of	PCT/US00/30040	11/01/00
PCT/US00/30040	Non-provisional of	60/215,130	06/30/00
PCT/US00/30040	Non-provisional of	60/163,580	11/05/99
10/100,683	Continuation-in-part of	PCT/US00/30037	11/01/00
PCT/US00/30037	Non-provisional of	60/215,137	06/30/00
PCT/US00/30037	Non-provisional of	60/163,577	11/05/99
10/100,683	Continuation-in-part of	PCT/US00/30045	11/01/00
PCT/US00/30045	Non-provisional of	60/215,133	06/30/00
PCT/US00/30045	Non-provisional of	60/163,581	11/05/99
10/100,683	Continuation-in-part of	PCT/US00/30036	11/01/00
PCT/US00/30036	Non-provisional of	60/221,366	07/27/00
PCT/US00/30036	Non-provisional of	60/163,576	11/05/99
10/100,683	Continuation-in-part of	PCT/US00/30039	11/01/00
PCT/US00/30039	Non-provisional of	60/221,367	07/27/00
PCT/US00/30039	Non-provisional of	60/195,296	04/07/00
PCT/US00/30039	Non-provisional of	60/164,344	11/09/99
10/100,683	Continuation-in-part of	PCT/US00/30654	11/08/00
PCT/US00/30654	Non-provisional of	60/221,142	07/27/00
PCT/US00/30654	Non-provisional of	60/164,835	11/12/99

10/100,683	Continuation-in-part of	PCT/US00/30628	11/08/00
PCT/US00/30628	Non-provisional of	60/215,140	06/30/00
PCT/US00/30628	Non-provisional of	60/164,744	11/12/99
10/100,683	Continuation-in-part of	PCT/US00/30653	11/08/00
PCT/US00/30653	Non-provisional of	60/221,193	07/27/00
PCT/US00/30653	Non-provisional of	60/164,735	11/12/99
10/100,683	Continuation-in-part of	PCT/US00/30629	11/08/00
PCT/US00/30629	Non-provisional of	60/222,904	08/03/00
PCT/US00/30629	Non-provisional of	60/164,825	11/12/99
10/100,683	Continuation-in-part of	PCT/US00/30679	11/08/00
PCT/US00/30679	Non-provisional of	60/224,007	08/04/00
PCT/US00/30679	Non-provisional of	60/164,834	11/12/99
10/100,683	Continuation-in-part of	PCT/US00/30674	11/08/00
PCT/US00/30674	Non-provisional of	60/215,128	06/30/00
PCT/US00/30674	Non-provisional of	60/164,750	11/12/99
10/100,683	Continuation-in-part of	PCT/US00/31162	11/15/00
60/215,136	Non-provisional of	60/215,136	06/30/00
60/215,136	Non-provisional of	60/166,415	11/19/99
10/100,683	Continuation-in-part of	PCT/US00/31282	11/15/00
PCT/US00/31282	Non-provisional of	60/219,665	07/21/00
PCT/US00/31282	Non-provisional of	60/166,414	11/19/99
10/100,683	Continuation-in-part of	PCT/US00/30657	11/08/00
PCT/US00/30657	Non-provisional of	60/215,132	06/30/00
PCT/US00/30657	Non-provisional of	60/164,731	11/12/99
10/100,683	Continuation-in-part of	PCT/US01/01396	01/17/01
60/256,968	Non-provisional of	60/256,968	12/21/00
60/256,968	Non-provisional of	60/226,280	08/18/00
10/100,683	Continuation-in-part of	PCT/US01/01387	01/17/01
60/259,803	Non-provisional of	60/259,803	01/05/01
60/259,803	Non-provisional of	60/226,380	08/18/00
10/100,683	Continuation-in-part of	PCT/US01/01567	01/17/01
PCT/US01/01567	Non-provisional of	60/228,084	08/28/00
10/100,683	Continuation-in-part of	PCT/US01/01431	01/17/01
PCT/US01/01431	Non-provisional of	60/231,968	09/12/00
PCT/US01/01431	Continuation-in-part of	09/915,582	07/27/01
10/100,683	Continuation-in-part of	PCT/US01/01432	01/17/01
PCT/US01/01432	Non-provisional of	60/236,326	09/29/00
10/100,683	Continuation-in-part of	PCT/US01/00544	01/09/01
PCT/US01/00544	Non-provisional of	60/234,211	09/20/00
10/100,683	Continuation-in-part of	PCT/US01/01435	01/17/01
PCT/US01/01435	Non-provisional of	60/226,282	08/18/00
10/100,683	Continuation-in-part of	PCT/US01/01386	01/17/01
PCT/US01/01386	Non-provisional of	60/232,104	09/12/00
10/100,683	Continuation-in-part of	PCT/US01/01565	01/17/01
PCT/US01/01565	Non-provisional of	60/234,210	09/20/00
10/100,683	Continuation-in-part of	PCT/US01/01394	01/17/01
PCT/US01/01394	Non-provisional of	60/259,805	01/05/01
PCT/US01/01394	Non-provisional of	60/226,278	08/18/00
10/100,683	Continuation-in-part of	PCT/US01/01434	01/17/01
PCT/US01/01434	Non-provisional of	60/259,678	01/05/01

PCT/US01/01434	Non-provisional of	60/226,279	08/18/00
10/100,683	Continuation-in-part of	PCT/US01/01397	01/17/01
PCT/US01/01397	Non-provisional of	60/226,281	08/18/00
10/100,683	Continuation-in-part of	PCT/US01/01385	01/17/01
PCT/US01/01385	Non-provisional of	60/231,969	09/12/00
10/100,683	Continuation-in-part of	PCT/US01/01384	01/17/01
PCT/US01/01384	Non-provisional of	60/259,516	01/04/01
PCT/US01/01384	Non-provisional of	60/228,086	08/28/00
10/100,683	Continuation-in-part of	PCT/US01/01383	01/17/01
PCT/US01/01383	Non-provisional of	60/259,804	01/05/01
PCT/US01/01383	Non-provisional of	60/228,083	08/28/00
10/100,683	Continuation-in-part of	PCT/US02/05064	02/21/02
PCT/US02/05064	Non-provisional of	60/304,444	07/12/01
PCT/US02/05064	Non-provisional of	60/270,658	02/23/01
10/100,683	Continuation-in-part of	PCT/US02/05301	02/21/02
PCT/US02/05301	Non-provisional of	60/304,417	07/12/01
PCT/US02/05301	Non-provisional of	60/270,625	02/23/01
10/100,683	Non-provisional of	60/304,121	07/11/01
10/100,683	Non-provisional of	60/295,869	06/06/01
10/100,683	Non-provisional of	60/325,209	09/28/01
10/100,683	Non-provisional of	60/311,085	08/10/01
10/100,683	Non-provisional of	60/330,629	10/26/01
10/100,683	Non-provisional of	60/331,046	11/07/01
10/100,683	Non-provisional of	60/358,554	02/22/02
10/100,683	Non-provisional of	60/358,714	02/25/02

; wherein each of the above applications are all herein incorporated by reference in their entirety.

5

Field of the Invention

The present invention relates to human secreted proteins/polypeptides, and isolated nucleic acid molecules encoding said proteins/polypeptides, useful for detecting, preventing, diagnosing, prognosticating, treating, and/or ameliorating allergic and asthmatic diseases and disorders. Antibodies that bind these polypeptides are also encompassed by the present invention. Also encompassed by the invention are vectors, host cells, and recombinant and synthetic methods for producing said polynucleotides, polypeptides, and/or antibodies. The invention further encompasses screening methods for identifying agonists and antagonists of polynucleotides and polypeptides of the invention. The present invention further encompasses methods and compositions for inhibiting or enhancing the production and function of the polypeptides of the present invention.

Background of the Invention

The immune system is an intricate network of cells, tissues and soluble molecules that function to protect the body from invasion by foreign substances and pathogens. The major cells of the immune system are lymphocytes, including B cells and T cells, and myeloid cells, including
5 basophils, eosinophils, neutrophils, mast cells, monocytes, macrophages and dendritic cells. In addition to these cellular components of the immune system, soluble molecules- such as antibodies, complement proteins, and cytokines- circulate in lymph and blood plasma, and play important roles in immunity.

The immune system can be subdivided into the acquired and innate immune systems. The
10 cells of the innate immune system (e.g., neutrophils, eosinophils, basophils, mast cells) are not antigen specific and their action is not enhanced by repeated exposure to the same antigen. The cells of the acquired immune system (B and T cells) are antigen specific. Repeated exposure of B and T cells to an antigen results in improved immune responses (memory responses) produced by these cell types. The cells and products of the acquired immune system can recruit components of
15 the innate system to mount a focused immune response. For a more extensive review of the immune system, see Fundamental Immunology, 4th edition, Ed. William Paul, Lippincott-Raven Pub. (1998).

An immune response is seldom carried out by a single cell type, but rather requires the coordinated efforts of several cell types. In order to coordinate an immune response, it is
20 necessary that cells of the immune system communicate with each other and with other cells of the body. Communication between cells may be made by cell-cell contact, between membrane bound molecules on each cell, or by the interaction of soluble components of the immune system with cellular receptors. Signaling between cell types may have one or more of a variety of consequences, including activation, proliferation, differentiation, and apoptosis. Activation and
25 differentiation of immune cells may result in the expression or secretion of polypeptides, or other molecules, which in turn affect the function of other cells and/or molecules of the immune system.

The genes and proteins associated with this coordinated immune response are essential for the proper regulation and functioning of the immune system. Dysregulation of immune system-related genes and proteins may result in a variety of diseases and/or disorders, including immediate
30 hypersensitivity diseases. Immediate hypersensitivity diseases, such as asthma, hay fever, and allergic conjunctivitis, are characterized by similar physiological mechanisms and generally are initiated by environmental antigens (e.g. pollen, dust, or molds). Patients suffering from the effects of these disorders are predisposed to react to specific external antigens. When these antigens contact certain tissues, such as ocular, nasal, or lung tissues, those tissues initiate an
35 immune response and produce undesirable and frequently life-threatening symptoms. Over 35

million Americans suffer from allergic disorders, such as seasonal allergic rhinitis (hay fever), and asthma affects about 10 million Americans. These conditions are not only becoming more common but also more serious, with more people being hospitalized.

5 Molecules that stimulate or suppress immune system function are known as immunomodulators. These molecules, which include endogenous proteins (e.g., cytokines, cytokine receptors, and intracellular signal transduction molecules), molecules derived from microorganisms, and synthetic agents, may exert their modulatory effects at one or more stages of the immune response, such as antigen recognition, stimulation of cytokine production and release, and/or activation/differentiation of lymphocytes and myeloid cells. Immunomodulators may
10 enhance (immunoprophylaxis, immunostimulation), restore (immunosubstitution, immunorestitution) or suppress (immunosuppression, immunodeviation) immunological functions or activities.

Immunomodulatory compounds have many important applications in clinical practice. For example, immunosuppressing agents (which attenuate or prevent unwanted immune
15 responses) can be used to prevent immediate hypersensitivity reactions such as asthma and allergic reactions. A mechanism of action common to many immunosuppressants is the inhibition of T cell activation and/or differentiation. Antilymphocyte antibodies have also been used to attenuate immune system functions. Currently used immunosuppressive agents can produce a number of side effects, which limit their use. Among the most serious secondary effects include kidney and
20 liver toxicity, increased risk of infection, hyperglycemia, neoplasia, and osteoporosis (see, e.g., Freeman, Clin. Biochem. 24(1):9-14 (1991); Mitchison, Dig. Dis. 11(2):78-101 (1993)). The discovery of new human allergy and/or asthma related polynucleotides, the polypeptides encoded by them, and antibodies that specifically bind these polypeptides, satisfies a need in the art by providing new compositions that are useful in the diagnosis, treatment, prevention and/or
25 prognosis of disorders of the immune system, including, but not limited to, allergic reactions and conditions, asthma, and related immediate hypersensitivity disorders.

Summary of the Invention

30 The present invention encompasses human secreted proteins/polypeptides, and isolated nucleic acid molecules encoding said proteins/polypeptides, useful for detecting, preventing, diagnosing, prognosticating, treating, and/or ameliorating allergic and asthmatic diseases and disorders. Antibodies that bind these polypeptides are also encompassed by the present invention; as are vectors, host cells, and recombinant and synthetic methods for producing said
35 polynucleotides, polypeptides, and/or antibodies. The invention further encompasses screening

methods for identifying agonists and antagonists of polynucleotides and polypeptides of the invention. The present invention also encompasses methods and compositions for inhibiting or enhancing the production and function of the polypeptides of the present invention.

5

Detailed Description

Polynucleotides and Polypeptides of the Invention

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Description of Table 1A

Table 1A summarizes information concerning certain polynucleotides and polypeptides of the invention. The first column provides the gene number in the application for each clone identifier. The second column provides a unique clone identifier, "Clone ID:", for a cDNA clone related to each contig sequence disclosed in Table 1A. Third column, the cDNA Clones identified in the second column were deposited as indicated in the third column (i.e. by ATCC Deposit No:Z and deposit date). Some of the deposits contain multiple different clones corresponding to the same gene. In the fourth column, "Vector" refers to the type of vector contained in the corresponding cDNA Clone identified in the second column. In the fifth column, the nucleotide sequence identified as "NT SEQ ID NO:X" was assembled from partially homologous ("overlapping") sequences obtained from the corresponding cDNA clone identified in the second column and, in some cases, from additional related cDNA clones. The overlapping sequences were assembled into a single contiguous sequence of high redundancy (usually three to five overlapping sequences at each nucleotide position), resulting in a final sequence identified as SEQ ID NO:X. In the sixth column, "Total NT Seq." refers to the total number of nucleotides in the contig sequence identified as SEQ ID NO:X." The deposited clone may contain all or most of these sequences, reflected by the nucleotide position indicated as "5' NT of Clone Seq." (seventh column) and the "3' NT of Clone Seq." (eighth column) of SEQ ID NO:X. In the ninth column, the nucleotide position of SEQ ID NO:X of the putative start codon (methionine) is identified as "5' NT of Start Codon." Similarly, in column ten, the nucleotide position of SEQ ID NO:X of the predicted signal sequence is identified as "5' NT of First AA of Signal Pep." In the eleventh column, the translated amino acid sequence, beginning with the methionine, is identified as "AA SEQ ID NO:Y," although other reading frames can also be routinely translated using known molecular biology techniques. The polypeptides produced by these alternative open reading frames are specifically contemplated by the present invention.

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In the twelfth and thirteenth columns of Table 1A, the first and last amino acid position of SEQ ID NO:Y of the predicted signal peptide is identified as "First AA of Sig Pep" and "Last AA of Sig Pep." In the fourteenth column, the predicted first amino acid position of SEQ ID NO:Y of the secreted portion is identified as "Predicted First AA of Secreted Portion". The amino acid position of SEQ ID NO:Y of the last amino acid encoded by the open reading frame is identified in the fifteenth column as "Last AA of ORF".

SEQ ID NO:X (where X may be any of the polynucleotide sequences disclosed in the sequence listing) and the translated SEQ ID NO:Y (where Y may be any of the polypeptide sequences disclosed in the sequence listing) are sufficiently accurate and otherwise suitable for a variety of uses well known in the art and described further below. For instance, SEQ ID NO:X is useful for designing nucleic acid hybridization probes that will detect nucleic acid sequences contained in SEQ ID NO:X or the cDNA contained in the deposited clone. These probes will also hybridize to nucleic acid molecules in biological samples, thereby enabling a variety of forensic and diagnostic methods of the invention. Similarly, polypeptides identified from SEQ ID NO:Y may be used, for example, to generate antibodies which bind specifically to proteins containing the polypeptides and the secreted proteins encoded by the cDNA clones identified in Table 1A and/or elsewhere herein

Nevertheless, DNA sequences generated by sequencing reactions can contain sequencing errors. The errors exist as misidentified nucleotides, or as insertions or deletions of nucleotides in the generated DNA sequence. The erroneously inserted or deleted nucleotides cause frame shifts in the reading frames of the predicted amino acid sequence. In these cases, the predicted amino acid sequence diverges from the actual amino acid sequence, even though the generated DNA sequence may be greater than 99.9% identical to the actual DNA sequence (for example, one base insertion or deletion in an open reading frame of over 1000 bases).

Accordingly, for those applications requiring precision in the nucleotide sequence or the amino acid sequence, the present invention provides not only the generated nucleotide sequence identified as SEQ ID NO:X, and the predicted translated amino acid sequence identified as SEQ ID NO:Y, but also a sample of plasmid DNA containing a human cDNA of the invention deposited with the ATCC, as set forth in Table 1A. The nucleotide sequence of each deposited plasmid can readily be determined by sequencing the deposited plasmid in accordance with known methods

The predicted amino acid sequence can then be verified from such deposits. Moreover, the amino acid sequence of the protein encoded by a particular plasmid can also be directly determined by peptide sequencing or by expressing the protein in a suitable host cell containing the deposited human cDNA, collecting the protein, and determining its sequence.

Also provided in Table 1A is the name of the vector which contains the cDNA plasmid. Each vector is routinely used in the art. The following additional information is provided for convenience.

Vectors Lambda Zap (U.S. Patent Nos. 5,128,256 and 5,286,636), Uni-Zap XR (U.S. Patent Nos. 5,128, 256 and 5,286,636), Zap Express (U.S. Patent Nos. 5,128,256 and 5,286,636), pBluescript (pBS) (Short, J. M. et al., *Nucleic Acids Res.* 16:7583-7600 (1988); Altling-Mees, M. A. and Short, J. M., *Nucleic Acids Res.* 17:9494 (1989)) and pBK (Altling-Mees, M. A. et al., *Strategies* 5:58-61 (1992)) are commercially available from Stratagene Cloning Systems, Inc., 11011 N. Torrey Pines Road, La Jolla, CA, 92037. pBS contains an ampicillin resistance gene and pBK contains a neomycin resistance gene. Phagemid pBS may be excised from the Lambda Zap and Uni-Zap XR vectors, and phagemid pBK may be excised from the Zap Express vector. Both phagemids may be transformed into *E. coli* strain XL-1 Blue, also available from Stratagene

Vectors pSport1, pCMVSPORT 1.0, pCMVSPORT 2.0 and pCMVSPORT 3.0, were obtained from Life Technologies, Inc., P. O. Box 6009, Gaithersburg, MD 20897. All Sport vectors contain an ampicillin resistance gene and may be transformed into *E. coli* strain DH10B, also available from Life Technologies. See, for instance, Gruber, C. E., et al., *Focus* 15:59 (1993). Vector lacmid BA (Bento Soares, Columbia University, New York, NY) contains an ampicillin resistance gene and can be transformed into *E. coli* strain XL-1 Blue. Vector pCR[®]2.1, which is available from Invitrogen, 1600 Faraday Avenue, Carlsbad, CA 92008, contains an ampicillin resistance gene and may be transformed into *E. coli* strain DH10B, available from Life Technologies. See, for instance, Clark, J. M., *Nuc. Acids Res.* 16:9677-9686 (1988) and Mead, D. et al., *Bio/Technology* 9: (1991).

The present invention also relates to the genes corresponding to SEQ ID NO:X, SEQ ID NO:Y, and/or a deposited cDNA (cDNA Clone ID). The corresponding gene can be isolated in accordance with known methods using the sequence information disclosed herein. Such methods include, but are not limited to, preparing probes or primers from the disclosed sequence and identifying or amplifying the corresponding gene from appropriate sources of genomic material.

Also provided in the present invention are allelic variants, orthologs, and/or species homologs. Procedures known in the art can be used to obtain full-length genes, allelic variants, splice variants, full-length coding portions, orthologs, and/or species homologs of genes corresponding to SEQ ID NO:X and SEQ ID NO:Y using information from the sequences disclosed herein or the clones deposited with the ATCC. For example, allelic variants and/or species homologs may be isolated and identified by making suitable probes or primers from the sequences provided herein and screening a suitable nucleic acid source for allelic variants and/or the desired homologue.

The present invention provides a polynucleotide comprising, or alternatively consisting of, the nucleic acid sequence of SEQ ID NO:X and/or a cDNA contained in ATCC Deposit No.Z. The present invention also provides a polypeptide comprising, or alternatively, consisting of, the polypeptide sequence of SEQ ID NO:Y, a polypeptide encoded by SEQ ID NO:X, and/or a polypeptide encoded by a cDNA contained in ATCC deposit No.Z. Polynucleotides encoding a polypeptide comprising, or alternatively consisting of the polypeptide sequence of SEQ ID NO:Y, a polypeptide encoded by SEQ ID NO:X and/or a polypeptide encoded by the cDNA contained in ATCC Deposit No.Z, are also encompassed by the invention. The present invention further encompasses a polynucleotide comprising, or alternatively consisting of the complement of the nucleic acid sequence of SEQ ID NO:X, and/or the complement of the coding strand of the cDNA contained in ATCC Deposit No.Z.

Description of Table 1B (Comprised of Tables 1B.1 and 1B.2)

Table 1B.1 and Table 1B.2 summarize some of the polynucleotides encompassed by the invention (including cDNA clones related to the sequences (Clone ID:), contig sequences (contig identifier (Contig ID:) and contig nucleotide sequence identifiers (SEQ ID NO:X)) and further summarizes certain characteristics of these polynucleotides and the polypeptides encoded thereby. The first column of Tables 1B.1 and 1B.2 provide the gene numbers in the application for each clone identifier. The second column of Tables 1B.1 and 1B.2 provide unique clone identifiers, "Clone ID:", for cDNA clones related to each contig sequence disclosed in Table 1A and/or Table 1B. The third column of Tables 1B.1 and 1B.2 provide unique contig identifiers, "Contig ID:" for each of the contig sequences disclosed in these tables. The fourth column of Tables 1B.1 and 1B.2 provide the sequence identifiers, "SEQ ID NO:X", for each of the contig sequences disclosed in Table 1A and/or 1B.

Table 1B.1

The fifth column of Table 1B.1, "ORF (From-To)", provides the location (i.e., nucleotide position numbers) within the polynucleotide sequence of SEQ ID NO:X that delineates the preferred open reading frame (ORF) that encodes the amino acid sequence shown in the sequence listing and referenced in Table 1B.1 as SEQ ID NO:Y (column 6). Column 7 of Table 1B.1 lists residues comprising predicted epitopes contained in the polypeptides encoded by each of the preferred ORFs (SEQ ID NO:Y). Identification of potential immunogenic regions was performed according to the method of Jameson and Wolf (CABIOS, 4; 181-186 (1988)); specifically, the Genetics Computer Group (GCG) implementation of this algorithm, embodied in the program PEPTIDESTRUCTURE (Wisconsin Package v10.0, Genetics Computer Group (GCG), Madison, Wisc.). This method returns a measure of the probability that a given residue is

found on the surface of the protein. Regions where the antigenic index score is greater than 0.9 over at least 6 amino acids are indicated in Table 1B.1 as "Predicted Epitopes". In particular embodiments, polypeptides of the invention comprise, or alternatively consist of, one, two, three, four, five or more of the predicted epitopes described in Table 1B.1. It will be appreciated that depending on the analytical criteria used to predict antigenic determinants, the exact address of the determinant may vary slightly. Column 8 of Table 1B.1 ("Tissue Distribution") is described below in Table 1B.2 Column 5. Column 9 of Table 1B.1 ("Cytologic Band") provides the chromosomal location of polynucleotides corresponding to SEQ ID NO:X. Chromosomal location was determined by finding exact matches to EST and cDNA sequences contained in the NCBI (National Center for Biotechnology Information) UniGene database. Given a presumptive chromosomal location, disease locus association was determined by comparison with the Morbid Map, derived from Online Mendelian Inheritance in Man (Online Mendelian Inheritance in Man, OMIM™. McKusick-Nathans Institute for Genetic Medicine, Johns Hopkins University (Baltimore, MD) and National Center for Biotechnology Information, National Library of Medicine (Bethesda, MD) 2000. World Wide Web URL: <http://www.ncbi.nlm.nih.gov/omim/>). If the putative chromosomal location of the Query overlaps with the chromosomal location of a Morbid Map entry, an OMIM identification number is disclosed in Table 1B.1, column 9 labeled "OMIM Disease Reference(s)". A key to the OMIM reference identification numbers is provided in Table 5.

Table 1B.2

Column 5 of Table 1B.2, "Tissue Distribution" shows the expression profile of tissue, cells, and/or cell line libraries which express the polynucleotides of the invention. The first code number shown in Table 1B.2 column 5 (preceding the colon), represents the tissue/cell source identifier code corresponding to the key provided in Table 4. Expression of these polynucleotides was not observed in the other tissues and/or cell libraries tested. The second number in column 5 (following the colon), represents the number of times a sequence corresponding to the reference polynucleotide sequence (e.g., SEQ ID NO:X) was identified in the corresponding tissue/cell source. Those tissue/cell source identifier codes in which the first two letters are "AR" designate information generated using DNA array technology. Utilizing this technology, cDNAs were amplified by PCR and then transferred, in duplicate, onto the array. Gene expression was assayed through hybridization of first strand cDNA probes to the DNA array. cDNA probes were generated from total RNA extracted from a variety of different tissues and cell lines. Probe synthesis was performed in the presence of ³³P dCTP, using oligo(dT) to prime reverse transcription. After hybridization, high stringency washing conditions were employed to remove non-specific hybrids from the array. The remaining signal, emanating from each gene target, was measured using a

Phosphorimager. Gene expression was reported as Phosphor Stimulating Luminescence (PSL) which reflects the level of phosphor signal generated from the probe hybridized to each of the gene targets represented on the array. A local background signal subtraction was performed before the total signal generated from each array was used to normalize gene expression between the different hybridizations. The value presented after “[array code]:” represents the mean of the duplicate values, following background subtraction and probe normalization. One of skill in the art could routinely use this information to identify normal and/or diseased tissue(s) which show a predominant expression pattern of the corresponding polynucleotide of the invention or to identify polynucleotides which show predominant and/or specific tissue and/or cell expression.

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Description of Table 1C

Table 1C summarizes additional polynucleotides encompassed by the invention (including cDNA clones related to the sequences (Clone ID:), contig sequences (contig identifier (Contig ID:) contig nucleotide sequence identifiers (SEQ ID NO:X)), and genomic sequences (SEQ ID NO:B). The first column provides a unique clone identifier, “Clone ID:”, for a cDNA clone related to each contig sequence. The second column provides the sequence identifier, “SEQ ID NO:X”, for each contig sequence. The third column provides a unique contig identifier, “Contig ID:” for each contig sequence. The fourth column, provides a BAC identifier “BAC ID NO:A” for the BAC clone referenced in the corresponding row of the table. The fifth column provides the nucleotide sequence identifier, “SEQ ID NO:B” for a fragment of the BAC clone identified in column four of the corresponding row of the table. The sixth column, “Exon From-To”, provides the location (i.e., nucleotide position numbers) within the polynucleotide sequence of SEQ ID NO:B which delineate certain polynucleotides of the invention that are also exemplary members of polynucleotide sequences that encode polypeptides of the invention (e.g., polypeptides containing amino acid sequences encoded by the polynucleotide sequences delineated in column six, and fragments and variants thereof).

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Description of Table 1D

Table 1D: In preferred embodiments, the present invention encompasses a method of detecting, preventing, diagnosing, prognosticating, treating, and/or ameliorating allergic and/or asthmatic diseases and disorders; comprising administering to a patient in which such treatment, prevention, or amelioration is desired a protein, nucleic acid, or antibody of the invention (or fragment or variant thereof) represented by Table 1A, Table 1B, and Table 1C, in an amount effective to detect, prevent, diagnose, prognosticate, treat, and/or ameliorate the disease or disorder.

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As indicated in Table 1D, the polynucleotides, polypeptides, agonists, or antagonists of the present invention (including antibodies) can be used in assays to test for one or more biological activities. If these polynucleotides and polypeptides do exhibit activity in a particular assay, it is likely that these molecules may be involved in the diseases associated with the biological activity.

5 Thus, the polynucleotides or polypeptides, or agonists or antagonists thereof (including antibodies) could be used to treat the associated disease.

Table 1D provides information related to biological activities for polynucleotides and polypeptides of the invention (including antibodies, agonists, and/or antagonists thereof). Table 1D also provides information related to assays which may be used to test polynucleotides and polypeptides of the invention (including antibodies, agonists, and/or antagonists thereof) for the corresponding biological activities. The first column ("Gene No.") provides the gene number in the application for each clone identifier. The second column ("cDNA Clone ID:") provides the unique clone identifier for each clone as previously described and indicated in Tables 1A, 1B, and 1C. The third column ("AA SEQ ID NO:Y") indicates the Sequence Listing SEQ ID Number for polypeptide sequences encoded by the corresponding cDNA clones (also as indicated in Tables 1A, 1B, and 2). The fourth column ("Biological Activity") indicates a biological activity corresponding to the indicated polypeptides (or polynucleotides encoding said polypeptides). The fifth column ("Exemplary Activity Assay") further describes the corresponding biological activity and provides information pertaining to the various types of assays that may be performed to test, demonstrate, or quantify the corresponding biological activity. Table 1D describes the use of FMAT technology, *inter alia*, for testing or demonstrating various biological activities. Fluorometric microvolume assay technology (FMAT) is a fluorescence-based system that provides a means to perform nonradioactive cell- and bead-based assays to detect activation of cell signal transduction pathways. This technology was designed specifically for ligand binding and immunological assays. Using this technology, fluorescent cells or beads at the bottom of the well are detected as localized areas of concentrated fluorescence using a data processing system. Unbound fluorophore comprising the background signal is ignored, allowing for a wide variety of homogeneous assays. FMAT technology may be used for peptide ligand binding assays, immunofluorescence, apoptosis, cytotoxicity, and bead-based immunocapture assays. *See*, 30 Miraglia S et. al., "Homogeneous cell and bead based assays for highthroughput screening using fluorometric microvolume assay technology," Journal of Biomolecular Screening; 4:193-204 (1999). In particular, FMAT technology may be used to test, confirm, and/or identify the ability of polypeptides (including polypeptide fragments and variants) to activate signal transduction pathways. For example, FMAT technology may be used to test, confirm, and/or identify the 35 ability of polypeptides to upregulate production of immunomodulatory proteins (such as, for

example, interleukins, GM-CSF, Rantes, and Tumor Necrosis factors, as well as other cellular regulators (e.g. insulin)).

Table 1D also describes the use of kinase assays for testing, demonstrating, or quantifying biological activity. In this regard, the phosphorylation and de-phosphorylation of specific amino acid residues (e.g. Tyrosine, Serine, Threonine) on cell-signal transduction proteins provides a fast, reversible means for activation and de-activation of cellular signal transduction pathways. Moreover, cell signal transduction via phosphorylation/de-phosphorylation is crucial to the regulation of a wide variety of cellular processes (e.g. proliferation, differentiation, migration, apoptosis, etc.). Accordingly, kinase assays provide a powerful tool useful for testing, confirming, and/or identifying polypeptides (including polypeptide fragments and variants) that mediate cell signal transduction events via protein phosphorylation. See e.g., Forrer, P., Tamaskovic R., and Jaussi, R. "Enzyme-Linked Immunosorbent Assay for Measurement of JNK, ERK, and p38 Kinase Activities" *Biol. Chem.* 379(8-9): 1101-1110 (1998).

Description of Table 1E

Polynucleotides encoding polypeptides of the present invention can be used in assays to test for one or more biological activities. One such biological activity which may be tested includes the ability of polynucleotides and polypeptides of the invention to stimulate up-regulation or down-regulation of expression of particular genes and proteins. Hence, if polynucleotides and polypeptides of the present invention exhibit activity in altering particular gene and protein expression patterns, it is likely that these polynucleotides and polypeptides of the present invention may be involved in, or capable of effecting changes in, diseases associated with the altered gene and protein expression profiles. Hence, polynucleotides, polypeptides, or antibodies of the present invention could be used to treat said associated diseases.

TaqMan® assays may be performed to assess the ability of polynucleotides (and polypeptides they encode) to alter the expression pattern of particular "target" genes. TaqMan® reactions are performed to evaluate the ability of a test agent to induce or repress expression of specific genes in different cell types. TaqMan® gene expression quantification assays ("TaqMan® assays") are well known to, and routinely performed by, those of ordinary skill in the art. TaqMan® assays are performed in a two step reverse transcription / polymerase chain reaction (RT-PCR). In the first (RT) step, cDNA is reverse transcribed from total RNA samples using random hexamer primers. In the second (PCR) step, PCR products are synthesized from the cDNA using gene specific primers.

To quantify gene expression the Taqman® PCR reaction exploits the 5' nuclease activity of AmpliTaq Gold® DNA Polymerase to cleave a Taqman® probe (distinct from the primers)

during PCR. The Taqman® probe contains a reporter dye at the 5'-end of the probe and a quencher dye at the 3' end of the probe. When the probe is intact, the proximity of the reporter dye to the quencher dye results in suppression of the reporter fluorescence. During PCR, if the target of interest is present, the probe specifically anneals between the forward and reverse primer sites. AmpliTaq Fold DNA Polymerase then cleaves the probe between the reporter and quencher when the probe hybridizes to the target, resulting in increased fluorescence of the reporter (see Figure 2). Accumulation of PCR products is detected directly by monitoring the increase in fluorescence of the reporter dye.

After the probe fragments are displaced from the target, polymerization of the strand continues. The 3'-end of the probe is blocked to prevent extension of the probe during PCR. This process occurs in every cycle and does not interfere with the exponential accumulation of product. The increase in fluorescence signal is detected only if the target sequence is complementary to the probe and is amplified during PCR. Because of these requirements, any nonspecific amplification is not detected.

For test sample preparation, vector controls or constructs containing the coding sequence for the gene of interest are transfected into cells, such as for example 293T cells, and supernatants collected after 48 hours. For cell treatment and RNA isolation, multiple primary human cells or human cell lines are used; such cells may include but are not limited to, Normal Human Dermal Fibroblasts, Aortic Smooth Muscle, Human Umbilical Vein Endothelial Cells, HepG2, Daudi, Jurkat, U937, Caco, and THP-1 cell lines. Cells are plated in growth media and growth is arrested by culturing without media change for 3 days, or by switching cells to low serum media and incubating overnight. Cells are treated for 1, 6, or 24 hours with either vector control supernatant or sample supernatant (or purified/partially purified protein preparations in buffer). Total RNA is isolated; for example, by using Trizol extraction or by using the Ambion RNAqueous(TM)-4PCR RNA isolation system. Expression levels of multiple genes are analyzed using TAQMAN, and expression in the test sample is compared to control vector samples to identify genes induced or repressed. Each of the above described techniques are well known to, and routinely performed by, those of ordinary skill in the art.

Table 1E indicates particular disease classes and preferred indications for which polynucleotides, polypeptides, or antibodies of the present invention may be used in detecting, diagnosing, preventing, treating and/or ameliorating said diseases and disorders based on "target" gene expression patterns which may be up- or down-regulated by polynucleotides (and the encoded polypeptides) corresponding to each indicated cDNA Clone ID (shown in Table 1E, Column 2).

Thus, in preferred embodiments, the present invention encompasses a method of detecting, diagnosing, preventing, treating, and/or ameliorating a disease or disorder listed in the "Disease Class" and/or "Preferred Indication" columns of Table 1E; comprising administering to a patient in which such detection, diagnosis, prevention, or treatment is desired a protein, nucleic acid, or antibody of the invention (or fragment or variant thereof) in an amount effective to detect, diagnose, prevent, treat, or ameliorate the disease or disorder. The first and second columns of Table 1D show the "Gene No." and "cDNA Clone ID No.", respectively, indicating certain nucleic acids and proteins (or antibodies against the same) of the invention (including polynucleotide, polypeptide, and antibody fragments or variants thereof) that may be used in detecting, diagnosing, preventing, treating, or ameliorating the disease(s) or disorder(s) indicated in column 6 and as indicated in the corresponding row in the "Disease Class" or "Preferred Indication" Columns of Table 1E.

In another embodiment, the present invention also encompasses methods of detecting, diagnosing, preventing, treating, or ameliorating a disease or disorder listed in the "Disease Class" or "Preferred Indication" Columns of Table 1E; comprising administering to a patient combinations of the proteins, nucleic acids, or antibodies of the invention (or fragments or variants thereof), sharing similar indications as shown in the corresponding rows in the "Disease Class" or "Preferred Indication" Columns of Table 1E.

The "Disease Class" Column of Table 1E provides a categorized descriptive heading for diseases, disorders, and/or conditions (more fully described below) that may be detected, diagnosed, prevented, treated, or ameliorated by a protein, nucleic acid, or antibody of the invention (or fragment or variant thereof).

The "Preferred Indication" Column of Table 1E describes diseases, disorders, and/or conditions that may be detected, diagnosed, prevented, treated, or ameliorated by a protein, nucleic acid, or antibody of the invention (or fragment or variant thereof).

The "Cell Line" and "Exemplary Targets" Columns of Table 1E indicate particular cell lines and target genes, respectively, which may show altered gene expression patterns (i.e., up- or down-regulation of the indicated target gene) in Taqman assays, performed as described above, utilizing polynucleotides of the cDNA Clone ID shown in the corresponding row. Alteration of expression patterns of the indicated "Exemplary Target" genes is correlated with a particular "Disease Class" and/or "Preferred Indication" as shown in the corresponding row under the respective column headings.

The "Exemplary Accessions" Column indicates GenBank Accessions (available online through the National Center for Biotechnology Information (NCBI) at

<http://www.ncbi.nlm.nih.gov/>) which correspond to the "Exemplary Targets" shown in the adjacent row.

The recitation of "Cancer" in the "Disease Class" Column indicates that the corresponding nucleic acid and protein, or antibody against the same, of the invention (or fragment or variant thereof) may be used for example, to detect, diagnose, prevent, treat, and/or ameliorate neoplastic diseases and/or disorders (e.g., leukemias, cancers, etc., as described below under "Hyperproliferative Disorders").

The recitation of "Immune" in the "Disease Class" column indicates that the corresponding nucleic acid and protein, or antibody against the same, of the invention (or fragment or variant thereof), may be used for example, to detect, diagnose, prevent, treat, and/or ameliorate diseases and/or disorders relating to neoplastic diseases (e.g., as described below under "Hyperproliferative Disorders"), blood disorders (e.g., as described below under "Immune Activity" "Cardiovascular Disorders" and/or "Blood-Related Disorders"), and infections (e.g., as described below under "Infectious Disease").

The recitation of "Angiogenesis" in the "Disease Class" column indicates that the corresponding nucleic acid and protein, or antibody against the same, of the invention (or fragment or variant thereof), may be used for example, to detect, diagnose, treat, prevent, and/or ameliorate diseases and/or disorders relating to neoplastic diseases (e.g., as described below under "Hyperproliferative Disorders"), diseases and/or disorders of the cardiovascular system (e.g., as described below under "Cardiovascular Disorders"), diseases and/or disorders involving cellular and genetic abnormalities (e.g., as described below under "Diseases at the Cellular Level"), diseases and/or disorders involving angiogenesis (e.g., as described below under "Anti-Angiogenesis Activity"), to promote or inhibit cell or tissue regeneration (e.g., as described below under "Regeneration"), or to promote wound healing (e.g., as described below under "Wound Healing and Epithelial Cell Proliferation").

The recitation of "Diabetes" in the "Disease Class" column indicates that the corresponding nucleic acid and protein, or antibody against the same, of the invention (or fragment or variant thereof), may be used for example, to detect, diagnose, treat, prevent, and/or ameliorate diabetes (including diabetes mellitus types I and II), as well as diseases and/or disorders associated with, or consequential to, diabetes (e.g. as described below under "Endocrine Disorders," "Renal Disorders," and "Gastrointestinal Disorders").

Description of Table 2

Table 2 summarizes homology and features of some of the polypeptides of the invention.

The first column provides a unique clone identifier, "Clone ID:", corresponding to a cDNA clone

disclosed in Table 1A or Table 1B. The second column provides the unique contig identifier, "Contig ID:" corresponding to contigs in Table 1B and allowing for correlation with the information in Table 1B. The third column provides the sequence identifier, "SEQ ID NO:X", for the contig polynucleotide sequence. The fourth column provides the analysis method by which the homology/identity disclosed in the Table was determined. Comparisons were made between polypeptides encoded by the polynucleotides of the invention and either a non-redundant protein database (herein referred to as "NR"), or a database of protein families (herein referred to as "PFAM") as further described below. The fifth column provides a description of the PFAM/NR hit having a significant match to a polypeptide of the invention. Column six provides the accession number of the PFAM/NR hit disclosed in the fifth column. Column seven, "Score/Percent Identity", provides a quality score or the percent identity, of the hit disclosed in columns five and six. Columns 8 and 9, "NT From" and "NT To" respectively, delineate the polynucleotides in "SEQ ID NO:X" that encode a polypeptide having a significant match to the PFAM/NR database as disclosed in the fifth and sixth columns. In specific embodiments polypeptides of the invention comprise, or alternatively consist of, an amino acid sequence encoded by a polynucleotide in SEQ ID NO:X as delineated in columns 8 and 9, or fragments or variants thereof.

Description of Table 3

Table 3 provides polynucleotide sequences that may be disclaimed according to certain embodiments of the invention. The first column provides a unique clone identifier, "Clone ID", for a cDNA clone related to contig sequences disclosed in Table 1B. The second column provides the sequence identifier, "SEQ ID NO:X", for contig sequences disclosed in Table 1A and/or Table 1B. The third column provides the unique contig identifier, "Contig ID:", for contigs disclosed in Table 1B. The fourth column provides a unique integer 'a' where 'a' is any integer between 1 and the final nucleotide minus 15 of SEQ ID NO:X, and the fifth column provides a unique integer 'b' where 'b' is any integer between 15 and the final nucleotide of SEQ ID NO:X, where both a and b correspond to the positions of nucleotide residues shown in SEQ ID NO:X, and where b is greater than or equal to a + 14. For each of the polynucleotides shown as SEQ ID NO:X, the uniquely defined integers can be substituted into the general formula of a-b, and used to describe polynucleotides which may be preferably excluded from the invention. In certain embodiments, preferably excluded from the invention are at least one, two, three, four, five, ten, or more of the polynucleotide sequence(s) having the accession number(s) disclosed in the sixth column of this Table (including for example, published sequence in connection with a particular BAC clone). In further embodiments, preferably excluded from the invention are the specific polynucleotide

sequence(s) contained in the clones corresponding to at least one, two, three, four, five, ten, or more of the available material having the accession numbers identified in the sixth column of this Table (including for example, the actual sequence contained in an identified BAC clone).

5 **Description of Table 4**

Table 4 provides a key to the tissue/cell source identifier code disclosed in Table 1B.2, column 5. Column 1 of Table 4 provides the tissue/cell source identifier code disclosed in Table 1B.2, column 5. Columns 2-5 provide a description of the tissue or cell source. Note that "Description" and "Tissue" sources (i.e. columns 2 and 3) having the prefix "a_" indicates organs, tissues, or cells derived from "adult" sources. Codes corresponding to diseased tissues are indicated in column 6 with the word "disease." The use of the word "disease" in column 6 is non-limiting. The tissue or cell source may be specific (e.g. a neoplasm), or may be disease-associated (e.g., a tissue sample from a normal portion of a diseased organ). Furthermore, tissues and/or cells lacking the "disease" designation may still be derived from sources directly or indirectly involved in a disease state or disorder, and therefore may have a further utility in that disease state or disorder. In numerous cases where the tissue/cell source is a library, column 7 identifies the vector used to generate the library.

20 **Description of Table 5**

Table 5 provides a key to the OMIM reference identification numbers disclosed in Table 1B.1, column 9. OMIM reference identification numbers (Column 1) were derived from Online Mendelian Inheritance in Man (Online Mendelian Inheritance in Man, OMIM. McKusick-Nathans Institute for Genetic Medicine, Johns Hopkins University (Baltimore, MD) and National Center for Biotechnology Information, National Library of Medicine, (Bethesda, MD) 2000. World Wide Web URL: <http://www.ncbi.nlm.nih.gov/omim/>). Column 2 provides diseases associated with the cytologic band disclosed in Table 1B.1, column 8, as determined using the Morbid Map database.

30 **Description of Table 6**

Table 6 summarizes some of the ATCC Deposits, Deposit dates, and ATCC designation numbers of deposits made with the ATCC in connection with the present application. These deposits were made in addition to those described in the Table 1A.

35 **Description of Table 7**

Table 7 shows the cDNA libraries sequenced, and ATCC designation numbers and vector information relating to these cDNA libraries.

The first column shows the first four letters indicating the Library from which each library clone was derived. The second column indicates the catalogued tissue description for the corresponding libraries. The third column indicates the vector containing the corresponding clones. The fourth column shows the ATCC deposit designation for each library clone as indicated by the deposit information in Table 6.

Definitions

The following definitions are provided to facilitate understanding of certain terms used throughout this specification.

10 In the present invention, "isolated" refers to material removed from its original environment (e.g., the natural environment if it is naturally occurring), and thus is altered "by the hand of man" from its natural state. For example, an isolated polynucleotide could be part of a vector or a composition of matter, or could be contained within a cell, and still be "isolated" because that vector, composition of matter, or particular cell is not the original environment of the polynucleotide. The term "isolated" does not refer to genomic or cDNA libraries, whole cell total or mRNA preparations, genomic DNA preparations (including those separated by electrophoresis and transferred onto blots), sheared whole cell genomic DNA preparations or other compositions where the art demonstrates no distinguishing features of the polynucleotide/sequences of the present invention.

20 In the present invention, a "secreted" protein refers to those proteins capable of being directed to the ER, secretory vesicles, or the extracellular space as a result of a signal sequence, as well as those proteins released into the extracellular space without necessarily containing a signal sequence. If the secreted protein is released into the extracellular space, the secreted protein can undergo extracellular processing to produce a "mature" protein. Release into the extracellular space can occur by many mechanisms, including exocytosis and proteolytic cleavage.

25 As used herein, a "polynucleotide" refers to a molecule having a nucleic acid sequence encoding SEQ ID NO:Y or a fragment or variant thereof (e.g., the polypeptide delineated in columns fourteen and fifteen of Table 1A); a nucleic acid sequence contained in SEQ ID NO:X (as described in column 5 of Table 1A and/or column 3 of Table 1B) or the complement thereof; a cDNA sequence contained in Clone ID: (as described in column 2 of Table 1A and/or Table 1B and contained within a library deposited with the ATCC); a nucleotide sequence encoding the polypeptide encoded by a nucleotide sequence in SEQ ID NO:B as defined in column 6 (EXON From-To) of Table 1C or a fragment or variant thereof; or a nucleotide coding sequence in SEQ ID NO:B as defined in column 6 of Table 1C or the complement thereof. For example, the polynucleotide can contain the nucleotide sequence of the full-length cDNA sequence, including

the 5' and 3' untranslated sequences, the coding region, as well as fragments, epitopes, domains, and variants of the nucleic acid sequence. Moreover, as used herein, a "polypeptide" refers to a molecule having an amino acid sequence encoded by a polynucleotide of the invention as broadly defined (obviously excluding poly-Phenylalanine or poly-Lysine peptide sequences which result from translation of a polyA tail of a sequence corresponding to a cDNA).

In the present invention, "SEQ ID NO:X" was often generated by overlapping sequences contained in multiple clones (contig analysis). A representative clone containing all or most of the sequence for SEQ ID NO:X is deposited at Human Genome Sciences, Inc. (HGS) in a catalogued and archived library. As shown, for example, in column 2 of Table 1B, each clone is identified by a cDNA Clone ID (identifier generally referred to herein as Clone ID:). Each Clone ID is unique to an individual clone and the Clone ID is all the information needed to retrieve a given clone from the HGS library. Table 7 provides a list of the deposited cDNA libraries. One can use the Clone ID: to determine the library source by reference to Tables 6 and 7. Table 7 lists the deposited cDNA libraries by name and links each library to an ATCC Deposit. Library names contain four characters, for example, "HTWE." The name of a cDNA clone (Clone ID) isolated from that library begins with the same four characters, for example "HTWEP07". As mentioned below, Table 1A and/or Table 1B correlates the Clone ID names with SEQ ID NO:X. Thus, starting with an SEQ ID NO:X, one can use Tables 1A, 1B, 6, 7, and 9 to determine the corresponding Clone ID, which library it came from and which ATCC deposit the library is contained in. Furthermore, it is possible to retrieve a given cDNA clone from the source library by techniques known in the art and described elsewhere herein. The ATCC is located at 10801 University Boulevard, Manassas, Virginia 20110-2209, USA. The ATCC deposits were made pursuant to the terms of the Budapest Treaty on the international recognition of the deposit of microorganisms for the purposes of patent procedure.

In specific embodiments, the polynucleotides of the invention are at least 15, at least 30, at least 50, at least 100, at least 125, at least 500, or at least 1000 continuous nucleotides but are less than or equal to 300 kb, 200 kb, 100 kb, 50 kb, 15 kb, 10 kb, 7.5kb, 5 kb, 2.5 kb, 2.0 kb, or 1 kb, in length. In a further embodiment, polynucleotides of the invention comprise a portion of the coding sequences, as disclosed herein, but do not comprise all or a portion of any intron. In another embodiment, the polynucleotides comprising coding sequences do not contain coding sequences of a genomic flanking gene (i.e., 5' or 3' to the gene of interest in the genome). In other embodiments, the polynucleotides of the invention do not contain the coding sequence of more than 1000, 500, 250, 100, 50, 25, 20, 15, 10, 5, 4, 3, 2, or 1 genomic flanking gene(s).

A "polynucleotide" of the present invention also includes those polynucleotides capable of hybridizing, under stringent hybridization conditions, to sequences contained in SEQ ID NO:X, or

the complement thereof (e.g., the complement of any one, two, three, four, or more of the polynucleotide fragments described herein), the polynucleotide sequence delineated in columns 7 and 8 of Table 1A or the complement thereof, the polynucleotide sequence delineated in columns 8 and 9 of Table 2 or the complement thereof, and/or cDNA sequences contained in Clone ID:
5 (e.g., the complement of any one, two, three, four, or more of the polynucleotide fragments, or the cDNA clone within the pool of cDNA clones deposited with the ATCC, described herein), and/or the polynucleotide sequence delineated in column 6 of Table 1C or the complement thereof. "Stringent hybridization conditions" refers to an overnight incubation at 42 degree C in a solution comprising 50% formamide, 5x SSC (750 mM NaCl, 75 mM trisodium citrate), 50 mM sodium
10 phosphate (pH 7.6), 5x Denhardt's solution, 10% dextran sulfate, and 20 µg/ml denatured, sheared salmon sperm DNA, followed by washing the filters in 0.1x SSC at about 65 degree C.

Also contemplated are nucleic acid molecules that hybridize to the polynucleotides of the present invention at lower stringency hybridization conditions. Changes in the stringency of hybridization and signal detection are primarily accomplished through the manipulation of
15 formamide concentration (lower percentages of formamide result in lowered stringency); salt conditions, or temperature. For example, lower stringency conditions include an overnight incubation at 37 degree C in a solution comprising 6X SSPE (20X SSPE = 3M NaCl; 0.2M NaH₂PO₄; 0.02M EDTA, pH 7.4), 0.5% SDS, 30% formamide, 100 ug/ml salmon sperm blocking DNA; followed by washes at 50 degree C with 1XSSPE, 0.1% SDS. In addition, to achieve even
20 lower stringency, washes performed following stringent hybridization can be done at higher salt concentrations (e.g. 5X SSC).

Note that variations in the above conditions may be accomplished through the inclusion and/or substitution of alternate blocking reagents used to suppress background in hybridization experiments. Typical blocking reagents include Denhardt's reagent, BLOTTO, heparin, denatured
25 salmon sperm DNA, and commercially available proprietary formulations. The inclusion of specific blocking reagents may require modification of the hybridization conditions described above, due to problems with compatibility.

Of course, a polynucleotide which hybridizes only to polyA⁺ sequences (such as any 3' terminal polyA⁺ tract of a cDNA shown in the sequence listing), or to a complementary stretch of
30 T (or U) residues, would not be included in the definition of "polynucleotide," since such a polynucleotide would hybridize to any nucleic acid molecule containing a poly (A) stretch or the complement thereof (e.g., practically any double-stranded cDNA clone generated using oligo dT as a primer).

The polynucleotide of the present invention can be composed of any polyribonucleotide or
35 polydeoxribonucleotide, which may be unmodified RNA or DNA or modified RNA or DNA. For

example, polynucleotides can be composed of single- and double-stranded DNA, DNA that is a mixture of single- and double-stranded regions, single- and double-stranded RNA, and RNA that is mixture of single- and double-stranded regions, hybrid molecules comprising DNA and RNA that may be single-stranded or, more typically, double-stranded or a mixture of single- and double-stranded regions. In addition, the polynucleotide can be composed of triple-stranded regions comprising RNA or DNA or both RNA and DNA. A polynucleotide may also contain one or more modified bases or DNA or RNA backbones modified for stability or for other reasons. "Modified" bases include, for example, tritylated bases and unusual bases such as inosine. A variety of modifications can be made to DNA and RNA; thus, "polynucleotide" embraces chemically, enzymatically, or metabolically modified forms.

In specific embodiments, the polynucleotides of the invention are at least 15, at least 30, at least 50, at least 100, at least 125, at least 500, or at least 1000 continuous nucleotides but are less than or equal to 300 kb, 200 kb, 100 kb, 50 kb, 15 kb, 10 kb, 7.5kb, 5 kb, 2.5 kb, 2.0 kb, or 1 kb, in length. In a further embodiment, polynucleotides of the invention comprise a portion of the coding sequences, as disclosed herein, but do not comprise all or a portion of any intron. In another embodiment, the polynucleotides comprising coding sequences do not contain coding sequences of a genomic flanking gene (i.e., 5' or 3' to the gene of interest in the genome). In other embodiments, the polynucleotides of the invention do not contain the coding sequence of more than 1000, 500, 250, 100, 50, 25, 20, 15, 10, 5, 4, 3, 2, or 1 genomic flanking gene(s).

"SEQ ID NO:X" refers to a polynucleotide sequence described in column 5 of Table 1A, while "SEQ ID NO:Y" refers to a polypeptide sequence described in column 10 of Table 1A. SEQ ID NO:X is identified by an integer specified in column 6 of Table 1A. The polypeptide sequence SEQ ID NO:Y is a translated open reading frame (ORF) encoded by polynucleotide SEQ ID NO:X. The polynucleotide sequences are shown in the sequence listing immediately followed by all of the polypeptide sequences. Thus, a polypeptide sequence corresponding to polynucleotide sequence SEQ ID NO:2 is the first polypeptide sequence shown in the sequence listing. The second polypeptide sequence corresponds to the polynucleotide sequence shown as SEQ ID NO:3, and so on.

The polypeptide of the present invention can be composed of amino acids joined to each other by peptide bonds or modified peptide bonds, i.e., peptide isosteres, and may contain amino acids other than the 20 gene-encoded amino acids. The polypeptides may be modified by either natural processes, such as posttranslational processing, or by chemical modification techniques which are well known in the art. Such modifications are well described in basic texts and in more detailed monographs, as well as in a voluminous research literature. Modifications can occur anywhere in a polypeptide, including the peptide backbone, the amino acid side-chains and the

amino or carboxyl termini. It will be appreciated that the same type of modification may be present in the same or varying degrees at several sites in a given polypeptide. Also, a given polypeptide may contain many types of modifications. Polypeptides may be branched, for example, as a result of ubiquitination, and they may be cyclic, with or without branching. Cyclic, branched, and branched cyclic polypeptides may result from posttranslation natural processes or may be made by synthetic methods. Modifications include acetylation, acylation, ADP-ribosylation, amidation, covalent attachment of flavin, covalent attachment of a heme moiety, covalent attachment of a nucleotide or nucleotide derivative, covalent attachment of a lipid or lipid derivative, covalent attachment of phosphatidylinositol, cross-linking, cyclization, disulfide bond formation, demethylation, formation of covalent cross-links, formation of cysteine, formation of pyroglutamate, formylation, gamma-carboxylation, glycosylation, GPI anchor formation, hydroxylation, iodination, methylation, myristoylation, oxidation, pegylation, proteolytic processing, phosphorylation, prenylation, racemization, selenoylation, sulfation, transfer-RNA mediated addition of amino acids to proteins such as arginylation, and ubiquitination. (See, for instance, PROTEINS - STRUCTURE AND MOLECULAR PROPERTIES, 2nd Ed., T. E. Creighton, W. H. Freeman and Company, New York (1993); POSTTRANSLATIONAL COVALENT MODIFICATION OF PROTEINS, B. C. Johnson, Ed., Academic Press, New York, pgs. 1-12 (1983); Seifter et al., Meth. Enzymol. 182:626-646 (1990); Rattan et al., Ann. N.Y. Acad. Sci. 663:48-62 (1992)).

"SEQ ID NO:X" refers to a polynucleotide sequence described, for example, in Tables 1A, Table 1B, or Table 2, while "SEQ ID NO:Y" refers to a polypeptide sequence described in column 11 of Table 1A and or Table 1B. SEQ ID NO:X is identified by an integer specified in column 4 of Table 1B. The polypeptide sequence SEQ ID NO:Y is a translated open reading frame (ORF) encoded by polynucleotide SEQ ID NO:X. "Clone ID:" refers to a cDNA clone described in column 2 of Table 1A and/or 1B.

"A polypeptide having functional activity" refers to a polypeptide capable of displaying one or more known functional activities associated with a full-length (complete) protein. Such functional activities include, but are not limited to, biological activity (e.g. activity useful in treating, preventing and/or ameliorating allergic and/or asthmatic diseases and disorders), antigenicity (ability to bind [or compete with a polypeptide for binding] to an anti-polypeptide antibody), immunogenicity (ability to generate antibody which binds to a specific polypeptide of the invention), ability to form multimers with polypeptides of the invention, and ability to bind to a receptor or ligand for a polypeptide.

The polypeptides of the invention can be assayed for functional activity (e.g. biological activity) using or routinely modifying assays known in the art, as well as assays described herein.

Specifically, one of skill in the art may routinely assay secreted polypeptides (including fragments and variants) of the invention for activity using assays as described in the examples section below.

"A polypeptide having biological activity" refers to a polypeptide exhibiting activity similar to, but not necessarily identical to, an activity of a polypeptide of the present invention, including mature forms, as measured in a particular biological assay, with or without dose dependency. In the case where dose dependency does exist, it need not be identical to that of the polypeptide, but rather substantially similar to the dose-dependence in a given activity as compared to the polypeptide of the present invention (i.e., the candidate polypeptide will exhibit greater activity or not more than about 25-fold less and, preferably, not more than about tenfold less activity, and most preferably, not more than about three-fold less activity relative to the polypeptide of the present invention).

TABLES

Table 1A

Table 1A summarizes information concerning certain polynucleotides and polypeptides of the invention. The first column provides the gene number in the application for each clone identifier. The second column provides a unique clone identifier, "Clone ID:", for a cDNA clone related to each contig sequence disclosed in Table 1A. Third column, the cDNA Clones identified in the second column were deposited as indicated in the third column (i.e. by ATCC Deposit No:Z and deposit date). Some of the deposits contain multiple different clones corresponding to the same gene. In the fourth column, "Vector" refers to the type of vector contained in the corresponding cDNA Clone identified in the second column. In the fifth column, the nucleotide sequence identified as "NT SEQ ID NO:X" was assembled from partially homologous ("overlapping") sequences obtained from the corresponding cDNA clone identified in the second column and, in some cases, from additional related cDNA clones. The overlapping sequences were assembled into a single contiguous sequence of high redundancy (usually three to five overlapping sequences at each nucleotide position), resulting in a final sequence identified as SEQ ID NO:X. In the sixth column, "Total NT Seq." refers to the total number of nucleotides in the contig sequence identified as SEQ ID NO:X." The deposited clone may contain all or most of these sequences, reflected by the nucleotide position indicated as "5' NT of Clone Seq." (seventh column) and the "3' NT of Clone Seq." (eighth column) of SEQ ID NO:X. In the ninth column, the nucleotide position of SEQ ID NO:X of the putative start codon (methionine) is identified as "5' NT of Start Codon." Similarly, in column ten, the nucleotide position of SEQ ID NO:X of the predicted signal sequence is identified as "5' NT of First AA of Signal Pep." In the eleventh column, the translated amino acid sequence, beginning with the methionine, is identified as "AA

SEQ ID NO:Y,” although other reading frames can also be routinely translated using known molecular biology techniques. The polypeptides produced by these alternative open reading frames are specifically contemplated by the present invention.

In the twelfth and thirteenth columns of Table 1A, the first and last amino acid position of SEQ ID NO:Y of the predicted signal peptide is identified as “First AA of Sig Pep” and “Last AA of Sig Pep.” In the fourteenth column, the predicted first amino acid position of SEQ ID NO:Y of the secreted portion is identified as “Predicted First AA of Secreted Portion”. The amino acid position of SEQ ID NO:Y of the last amino acid encoded by the open reading frame is identified in the fifteenth column as “Last AA of ORF”.

SEQ ID NO:X (where X may be any of the polynucleotide sequences disclosed in the sequence listing) and the translated SEQ ID NO:Y (where Y may be any of the polypeptide sequences disclosed in the sequence listing) are sufficiently accurate and otherwise suitable for a variety of uses well known in the art and described further below. For instance, SEQ ID NO:X is useful for designing nucleic acid hybridization probes that will detect nucleic acid sequences contained in SEQ ID NO:X or the cDNA contained in the deposited clone. These probes will also hybridize to nucleic acid molecules in biological samples, thereby enabling a variety of forensic and diagnostic methods of the invention. Similarly, polypeptides identified from SEQ ID NO:Y may be used, for example, to generate antibodies which bind specifically to proteins containing the polypeptides and the secreted proteins encoded by the cDNA clones identified in Table 1A and/or elsewhere herein

Nevertheless, DNA sequences generated by sequencing reactions can contain sequencing errors. The errors exist as misidentified nucleotides, or as insertions or deletions of nucleotides in the generated DNA sequence. The erroneously inserted or deleted nucleotides cause frame shifts in the reading frames of the predicted amino acid sequence. In these cases, the predicted amino acid sequence diverges from the actual amino acid sequence, even though the generated DNA sequence may be greater than 99.9% identical to the actual DNA sequence (for example, one base insertion or deletion in an open reading frame of over 1000 bases).

Accordingly, for those applications requiring precision in the nucleotide sequence or the amino acid sequence, the present invention provides not only the generated nucleotide sequence identified as SEQ ID NO:X, and the predicted translated amino acid sequence identified as SEQ ID NO:Y, but also a sample of plasmid DNA containing a human cDNA of the invention deposited with the ATCC, as set forth in Table 1A. The nucleotide sequence of each deposited plasmid can readily be determined by sequencing the deposited plasmid in accordance with known methods

The predicted amino acid sequence can then be verified from such deposits. Moreover,

the amino acid sequence of the protein encoded by a particular plasmid can also be directly determined by peptide sequencing or by expressing the protein in a suitable host cell containing the deposited human cDNA, collecting the protein, and determining its sequence.

Also provided in Table 1A is the name of the vector which contains the cDNA plasmid.

5 Each vector is routinely used in the art. The following additional information is provided for convenience.

Vectors Lambda Zap (U.S. Patent Nos. 5,128,256 and 5,286,636), Uni-Zap XR (U.S. Patent Nos. 5,128, 256 and 5,286,636), Zap Express (U.S. Patent Nos. 5,128,256 and 5,286,636), pBluescript (pBS) (Short, J. M. et al., *Nucleic Acids Res.* 16:7583-7600 (1988); Altting-Mees, M. A. and Short, J. M., *Nucleic Acids Res.* 17:9494 (1989)) and pBK (Altting-Mees, M. A. et al., *Strategies* 5:58-61 (1992)) are commercially available from Stratagene Cloning Systems, Inc., 11011 N. Torrey Pines Road, La Jolla, CA, 92037. pBS contains an ampicillin resistance gene and pBK contains a neomycin resistance gene. Phagemid pBS may be excised from the Lambda Zap and Uni-Zap XR vectors, and phagemid pBK may be excised from the Zap Express vector. Both
10
15 phagemids may be transformed into *E. coli* strain XL-1 Blue, also available from Stratagene

Vectors pSport1, pCMVSport 1.0, pCMVSport 2.0 and pCMVSport 3.0, were obtained from Life Technologies, Inc., P. O. Box 6009, Gaithersburg, MD 20897. All Sport vectors contain an ampicillin resistance gene and may be transformed into *E. coli* strain DH10B, also available from Life Technologies. See, for instance, Gruber, C. E., et al., *Focus* 15:59 (1993). Vector
20 lafmid BA (Bento Soares, Columbia University, New York, NY) contains an ampicillin resistance gene and can be transformed into *E. coli* strain XL-1 Blue. Vector pCR[®]2.1, which is available from Invitrogen, 1600 Faraday Avenue, Carlsbad, CA 92008, contains an ampicillin resistance gene and may be transformed into *E. coli* strain DH10B, available from Life Technologies. See, for instance, Clark, J. M., *Nuc. Acids Res.* 16:9677-9686 (1988) and Mead, D. et al.,
25 *Bio/Technology* 9: (1991).

The present invention also relates to the genes corresponding to SEQ ID NO:X, SEQ ID NO:Y, and/or a deposited cDNA (cDNA Clone ID). The corresponding gene can be isolated in accordance with known methods using the sequence information disclosed herein. Such methods include, but are not limited to, preparing probes or primers from the disclosed sequence and
30 identifying or amplifying the corresponding gene from appropriate sources of genomic material.

Also provided in the present invention are allelic variants, orthologs, and/or species homologs. Procedures known in the art can be used to obtain full-length genes, allelic variants, splice variants, full-length coding portions, orthologs, and/or species homologs of genes corresponding to SEQ ID NO:X and SEQ ID NO:Y using information from the sequences
35 disclosed herein or the clones deposited with the ATCC. For example, allelic variants and/or

species homologs may be isolated and identified by making suitable probes or primers from the sequences provided herein and screening a suitable nucleic acid source for allelic variants and/or the desired homologue.

5 The present invention provides a polynucleotide comprising, or alternatively consisting of, the nucleic acid sequence of SEQ ID NO:X and/or a cDNA contained in ATCC Deposit No.Z. The present invention also provides a polypeptide comprising, or alternatively, consisting of, the polypeptide sequence of SEQ ID NO:Y, a polypeptide encoded by SEQ ID NO:X, and/or a polypeptide encoded by a cDNA contained in ATCC deposit No.Z. Polynucleotides encoding a polypeptide comprising, or alternatively consisting of the polypeptide sequence of SEQ ID NO:Y,
10 a polypeptide encoded by SEQ ID NO:X and/or a polypeptide encoded by the cDNA contained in ATCC Deposit No.Z, are also encompassed by the invention. The present invention further encompasses a polynucleotide comprising, or alternatively consisting of the complement of the nucleic acid sequence of SEQ ID NO:X, and/or the complement of the coding strand of the cDNA contained in ATCC Deposit No.Z.

15

TABLE 1A

Gene No.	cDNA Clone ID	ATCC Deposit No.:Z and Date	Vector	NT SEQ ID NO: X	Total NT Seq.	5' NT of Clone Seq.	3' NT of Clone Seq.	5' NT of Start Codon	5' NT of First AA of Signal Pep	AA SEQ ID NO: Y	First AA of Sig Pep	Last AA of Sig Pep	First AA of Secreted Portion	Last AA of ORF
1	H2MAC30	209299 09/25/97	pBluescript SK-	11	459	1	459	157	157	300	1	28	29	72
2	H6EDF66	209299 09/25/97	Uni-ZAP XR	12	540	1	540	146	146	301	1	27	28	131
3	H6EDX46	209626 02/12/98	Uni-ZAP XR	13	888	1	888	229	229	302	1	20	21	182
3	H6EDX46	209626 02/12/98	Uni-ZAP XR	203	718	1	718	128	128	492	1	20	21	84
4	HABAG37	209626 02/12/98	pSport1	14	654	1	639	97	97	303	1	31	32	62
5	HACBD91	209626 02/12/98	Uni-ZAP XR	15	1445	1	1445	117	117	304	1	42	43	49
6	HACCI17	203071 07/27/98	Uni-ZAP XR	16	1722	336	1714	461	461	305	1	24	25	218
6	HACCI17	203071 07/27/98	Uni-ZAP XR	204	1380	12	1380	135	135	493	1	24	25	72
7	HAGAM64	209603 01/29/98	Uni-ZAP XR	17	2321	1	2321	57	57	306	1	31	32	44
8	HAHDR32	209626 02/12/98	Uni-ZAP XR	18	1256	365	1256	435	435	307	1	25	26	181
9	HAIBO71	209145 07/17/97	Uni-ZAP XR	19	752	172	752	325	325	308	1	28	29	66

Gene No.	cDNA Clone ID	ATCC Deposit No.:Z and Date	Vector	NT SEQ ID NO: X	Total NT Seq.	5' NT of Clone Seq.	3' NT of Clone Seq.	5' NT of Start Codon	5' NT of First AA of Signal Pep	AA SEQ ID NO: Y	First AA of Sig Pep	Last AA of Sig Pep	First AA of Secreted Portion	Last AA of ORF
10	HAF57	203364 10/19/98	pCMVSPORT 3.0	20	2761	1	2761	43	43	309	1	1	2	94
11	HAMFC93	PTA-849 10/13/99	pCMVSPORT 3.0	21	2534	1	2534	136	136	310	1	30	31	191
11	HAMFC93	PTA-849 10/13/99	pCMVSPORT 3.0	205	824	1	824	115	115	494	1	30	31	178
11	HAMFC93	PTA-849 10/13/99	pCMVSPORT 3.0	206	3941	1947	3941		323	495	1			8
12	HAPNY86	209511 12/03/97	Uni-ZAP XR	22	1280	1	1280	100	100	311	1	25	26	129
13	HATDF29	203858 03/18/99	Uni-ZAP XR	23	1355	1	1355	143	143	312	1	30	31	385
14	HBAFJ33	209603 01/29/98	pSPORT1	24	1280	1	1252	60	60	313	1	15	16	110
15	HBAFV19	PTA-1543 03/21/00	pSPORT1	25	953	1	953	6	6	314	1	1	2	258
16	HBIBW67	209324 10/02/97	Uni-ZAP XR	26	1404	1	1404	685	685	315	1	33	34	38
17	HBIMB51	209683 03/20/98	pCMVSPORT 3.0	27	537	1	537	98	98	316	1	21	22	146
17	HBIMB51	209683 03/20/98	pCMVSPORT 3.0	207	526	1	526	93	93	496	1	21	22	130
18	HBID05	209300 09/25/97	Uni-ZAP XR	28	2008	1	2008	157	157	317	1	20	21	199
18	HBID05	209300 09/25/97	Uni-ZAP XR	208	571	1	571	137	137	497	1	20	21	111

Gene No.	cDNA Clone ID	ATCC Deposit No.:Z and Date	Vector	NT SEQ ID NO: X	Total NT Seq.	5' NT of Clone Seq.	3' NT of Clone Seq.	5' NT of Start Codon	5' NT of First AA of Signal Pep	AA SEQ ID NO: Y	First AA of Sig Pep	Last AA of Sig Pep	First AA of Secreted Portion	Last AA of ORF
19	HBJU28	209346 10/09/97	Uni-ZAP XR	29	1160	1	1160	133	133	318	1	18	19	84
20	HBJLH40	203499 12/01/98	Uni-ZAP XR	30	1853	1	1853	74	74	319	1	30	31	74
21	HBXFL29	203858 03/18/99	ZAP Express	31	2229	376	2210	560	560	320	1	31	32	57
22	HCACU58	209626 02/12/98	Uni-ZAP XR	32	1554	1	1554	137	137	321	1	30	31	83
23	HCDBW86	209242 09/12/97	Uni-ZAP XR	33	730	1	730	139	139	322	1	18	19	30
24	HCE3G69	209878 05/18/98	Uni-ZAP XR	34	2084	1	2084	165	165	323	1	19	20	336
24	HCE3G69	209878 05/18/98	Uni-ZAP XR	209	2078	1	2078	165	165	498	1	19	20	105
25	HCEEA88	209626 02/12/98	Uni-ZAP XR	35	1016	1	1016	134	134	324	1	23	24	60
26	HCEFB69	209965 06/11/98	Uni-ZAP XR	36	1430	1	1430	188	188	325	1	24	25	224
27	HCFMV71	209242 09/12/97	pSport1	37	400	1	400	31	31	326	1	24	25	58
28	HCNSD93	209627 02/12/98	pBluescript	38	1106	1	1106	139	139	327	1	21	22	46
29	HCUIM65	209324 10/02/97	ZAP Express	39	875	331	736	557	557	328	1	27	28	47
30	HCWKC15	209324 10/02/97	ZAP Express	40	710	1	710	37	37	329	1	18	19	40

Gene No.	cDNA Clone ID	ATCC Deposit No./Z and Date	Vector	NT SEQ ID NO: X	Total NT Seq.	5' NT of Clone Seq.	3' NT of Clone Seq.	5' NT of Start Codon	5' NT of First AA of Signal Pep	AA SEQ ID NO: Y	First AA of Sig Pep	Last AA of Sig Pep	First AA of Secreted Portion	Last AA of ORF
31	HCWLD74	209626 02/12/98	ZAP Express	41	1540	1	1540	138	138	330	1	21	22	65
32	HDHEB60	209215 08/21/97	pCMVSPORT 2.0	42	1421	235	1421	568	568	331	1	24	25	108
33	HDHMA45	203331 10/08/98	pCMVSPORT 2.0	43	2184	1	2184	199	199	332	1	33	34	413
33	HDHMA45	203331 10/08/98	pCMVSPORT 2.0	210	2190	1	2190	204	204	499	1	33	34	413
34	HDHMA72	209324 10/02/97	pCMVSPORT 2.0	44	4463	216	2158	287	287	333	1	36	37	315
35	HDPBA28	PTA-163 06/01/99	pCMVSPORT 3.0	45	3447	197	3447	259	259	334	1	32	33	941
35	HDPBA28	PTA-163 06/01/99	pCMVSPORT 3.0	211	4909	1	4909	69	69	500	1	32	33	941
36	HDPCO25	209125 06/19/97	pCMVSPORT 3.0	46	767	76	767	182	182	335	1	20	21	53
37	HDPCY37	209568 01/06/98	pCMVSPORT 3.0	47	1932	45	1932	76	76	336	1	21	22	578
37	HDPCY37	209568 01/06/98	pCMVSPORT 3.0	212	1931	45	1931	76	76	501	1	21	22	264
38	HDPH151	209125 06/19/97	pCMVSPORT 3.0	48	728	1	728	245	245	337	1	30	31	40
39	HDPND46	209627 02/12/98	pCMVSPORT 3.0	49	1727	1	1727	15	15	338	1	22	23	484
40	HDPOH06	209745 04/07/98	pCMVSPORT 3.0	50	2504	1	2504	252	252	339	1	29	30	242

Gene No.	cDNA Clone ID	ATCC Deposit No:Z and Date	Vector	NT SEQ ID NO: X	Total NT Seq.	5' NT of Clone Seq.	3' NT of Clone Seq.	5' NT of Start Codon	5' NT of First AA of Signal Pep	AA SEQ ID NO: Y	First AA of Sig Pep	Last AA of Sig Pep	First AA of Secreted Portion	Last AA of ORF
41	HDPSP54	209782 04/20/98	pCMVSPORT 3.0	51	3091	2304	3091	2356	2356	340	1	18	19	48
41	HDPSP54	209782 04/20/98	pCMVSPORT 3.0	213	536	1	536	179	179	502	1	41	42	55
42	HDPVH60	203105 08/13/98	pCMVSPORT 3.0	52	3116	1	3100	8	8	341	1	45	46	51
43	HDPWN93	PTA-868 10/26/99	pCMVSPORT 3.0	53	2679	1	2669	45	45	342	1	19	20	802
43	HDPWN93	PTA-868 10/26/99	pCMVSPORT 3.0	214	716	1	716	35	35	503	1	19	20	214
43	HDPWN93	PTA-868 10/26/99	pCMVSPORT 3.0	215	2716	26	2716	27	27	504	1	19	20	43
44	HDQHD03	203570 01/11/99	pCMVSPORT 3.0	54	1266	1	1266	274	274	343	1	20	21	331
44	HDQHD03	203570 01/11/99	pCMVSPORT 3.0	216	1257	1	1257	259	259	505	1	20	21	333
45	HE2EN04	209300 09/25/97	Uni-ZAP XR	55	370	1	370	57	57	344	1	16	17	50
46	HE8QV67	PTA-2072 06/09/00	Uni-ZAP XR	56	1999	643	1999	502	502	345	1	49	50	80
46	HE8QV67	PTA-2072 06/09/00	Uni-ZAP XR	217	2342	1956	2276		256	506	1	1	2	415
47	HE8UB86	203570 01/11/99	Uni-ZAP XR	57	1021	1	1021	201	201	346	1	21	22	250
48	HE9BK23	209683 03/20/98	Uni-ZAP XR	58	1636	1	1636	39	39	347	1	21	22	309

Gene No.	cDNA Clone ID	ATCC Deposit No:Z and Date	Vector	NT SEQ ID NO: X	Total NT Seq.	5' NT of Clone Seq.	3' NT of Clone Seq.	5' NT of Start Codon	5' NT of First AA of Signal Pep	AA SEQ ID NO: Y	First AA of Sig Pep	Last AA of Sig Pep	First AA of Secreted Portion	Last AA of ORF
49	HEBBN36	209141 07/09/97	Uni-ZAP XR	59	1046	470	1046	645	645	348	1	29	30	53
50	HEQCC55	209965 06/11/98	pCMVSPORT 3.0	60	1000	1	1000	25	25	349	1	27	28	129
50	HEQCC55	209965 06/11/98	pCMVSPORT 3.0	218	1052	30	1052	62	62	507	1	27	28	112
50	HEQCC55	209965 06/11/98	pCMVSPORT 3.0	219	1037	1	1037	57	57	508	1	27	28	155
51	HESAJ10	209242 09/12/97	Uni-ZAP XR	61	1090	400	1090	405	405	350	1	23	24	71
52	HETEU28	PTA-842 10/13/99	Uni-ZAP XR	62	1381	1	1381	256	256	351	1	34	35	153
52	HETEU28	PTA-842 10/13/99	Uni-ZAP XR	220	1501	1	1462	331	331	509	1	34	35	153
53	HFABG18	PTA-1544 03/21/00	Uni-ZAP XR	63	1345	1	1345	53	53	352	1	26	27	87
54	HFAMB72	209146 07/17/97	Uni-ZAP XR	64	1323	509	1323	559	559	353	1	22	23	60
55	HFCCQ50	209463 11/14/97	Uni-ZAP XR	65	1271	1	1271	47	47	354	1	20	21	352
56	HFIIJ70	PTA-846 10/13/99	pSPORT1	66	1408	1	1408	24	24	355	1	23	24	47
56	HFIIJ70	PTA-846 10/13/99	pSPORT1	221	1441	43	1441	74	74	510	1	23	24	47
57	HFKET18	PTA-622 09/02/99	Uni-ZAP XR	67	2407	1	2407	137	137	356	1	14	15	74

Gene No.	cDNA Clone ID	ATCC Deposit No:Z and Date	Vector	NT SEQ ID NO: X	Total NT Seq.	5' NT of Clone Seq.	3' NT of Clone Seq.	5' NT of Start Codon	5' NT of First AA of Signal Pep	AA SEQ ID NO: Y	First AA of Sig Pep	Last AA of Sig Pep	First AA of Secreted Portion	Last AA of ORF
58	HFLNB64	209463 11/14/97	Uni-ZAP XR	68	648	1	648	62	62	357	1	39	40	45
59	HFOXA73	209277 09/18/97	pSportI	69	540	1	540	25	25	358	1	17	18	52
59	HFOXA73	209277 09/18/97	pSportI	222	539	1	539	15	15	511	1			17
60	HFPAC12	209511 12/03/97	Uni-ZAP XR	70	1088	1	1088	140	140	359	1	21	22	88
61	HFP AO71	209626 02/12/98	Uni-ZAP XR	71	2067	364	2067	414	414	360	1	33	34	131
62	HFPCX36	209242 09/12/97	Uni-ZAP XR	72	796	1	796	103	103	361	1	27	28	46
63	HFPCX64	209878 05/18/98	Uni-ZAP XR	73	1076	1	1076	181	181	362	1	28	29	87
63	HFPCX64	209878 05/18/98	Uni-ZAP XR	223	1069	1	1069	181	181	512	1	28	29	181
63	HFPCX64	209878 05/18/98	Uni-ZAP XR	224	1154	84	1154	257	257	513	1	28	29	87
63	HFPCX64	209878 05/18/98	Uni-ZAP XR	225	1197	85	1197	257	257	514	1	28	29	87
64	HFTBM50	209300 09/25/97	Uni-ZAP XR	74	762	1	740	158	158	363	1	20	21	34
65	HFXDJ75	209603 01/29/98	Lambda ZAP II	75	1918	1	1914	44	44	364	1	26	27	41
66	HFXJU68	209423 10/30/97	Lambda ZAP II	76	712	1	712	141	141	365	1	26	27	162

Gene No.	cDNA Clone ID	ATCC Deposit No.:Z and Date	Vector	NT SEQ ID NO: X	Total NT Seq.	5' NT of Clone Seq.	3' NT of Clone Seq.	5' NT of Start Codon	5' NT of First AA of Signal Pep	AA SEQ ID NO: Y	First AA of Sig Pep	Last AA of Sig Pep	First AA of Secreted Portion	Last AA of ORF
66	HFXJU68	209423 10/30/97	Lambda ZAP II	226	1347	1	1347	148	148	515	1	25	26	66
67	HGBIB74	203648 02/09/99	Uni-ZAP XR	77	1816	1	1804	14	14	366	1	23	24	377
67	HGBIB74	203648 02/09/99	Uni-ZAP XR	227	1821	1	1821	28	28	516	1	20	21	170
67	HGBIB74	203648 02/09/99	Uni-ZAP XR	228	1094	1	1094		2	517	1	1	2	151
68	HHEMA75	209179 07/24/97	pCMVSPORT 3.0	78	865	229	865	569	569	367	1	35	36	84
69	HHENK42	209195 08/01/97	pCMVSPORT 3.0	79	656	1	656	63	63	368	1	7	8	42
70	HHEPM33	PTA-322 07/09/99	pCMVSPORT 3.0	80	1459	1	1459	269	269	369	1	20	21	82
71	HHEPT60	209138 07/03/97	pCMVSPORT 3.0	81	532	21	532	245	245	370	1	18	19	36
72	HHFHJ59	97975 04/04/97 209081 05/29/97	Uni-ZAP XR	82	661	1	661	192	192	371	1	29	30	112
73	HHGDW43	209346 10/09/97	Lambda ZAP II	83	1050	1	1050	107	107	372	1	40	41	44
74	HHPEC09	209877 05/18/98	Uni-ZAP XR	84	488	1	488	71	71	373	1	19	20	55
75	HHPTJ65	209179 07/24/97	Uni-ZAP XR	85	515	1	515	247	247	374	1	32	33	48

Gene No.	cDNA Clone ID	ATCC Deposit No:Z and Date	Vector	NT SEQ ID NO: X	Total NT Seq.	5' NT of Clone Seq.	3' NT of Clone Seq.	5' NT of Start Codon	5' NT of First AA of Signal Pep	AA SEQ ID NO: Y	First AA of Sig Pep	Last AA of Sig Pep	First AA of Secreted Portion	Last AA of ORF
76	HJABX32	209146 07/17/97	pBluescript SK-	86	1061	454	1061	557	557	375	1	18	19	51
77	HJBCU04	PTA-322 07/09/99	pBluescript SK-	87	1192	1	1192	96	96	376	1	49	50	176
78	HJMBN89	209407 10/23/97	pCMV/Sport 3.0	88	1064	306	1064	348	348	377	1	13	14	56
79	HJMBW30	209146 07/17/97	pCMV/Sport 3.0	89	884	1	874	110	110	378	1	18	19	42
80	HKABI84	209603 01/29/98	pCMV/Sport 2.0	90	1238	45	1238	274	274	379	1	16	17	47
81	HKACB56	209346 10/09/97	pCMV/Sport 2.0	91	496	1	496	27	27	380	1	23	24	80
82	HKACM93	PTA-849 10/13/99	pCMV/Sport 2.0	92	2352	1	2352	218	218	381	1	30	31	692
82	HKACM93	PTA-849 10/13/99	pCMV/Sport 2.0	229	549	1	549	189	189	518	1	30	31	120
82	HKACM93	PTA-849 10/13/99	pCMV/Sport 2.0	230	1120	1	1120	314	314	519	1	30	31	269
82	HKACM93	PTA-849 10/13/99	pCMV/Sport 2.0	231	1893	739	1893		202	520	1	13	14	17
82	HKACM93	PTA-849 10/13/99	pCMV/Sport 2.0	232	1187	1	1187		638	521	1	4	5	45
83	HKADQ91	209568 01/06/98	pCMV/Sport 2.0	93	1523	30	1517	229	229	382	1	25	26	275
84	HKAEG43	209965 06/11/98	pCMV/Sport 2.0	94	1297	1	1297	32	32	383	1	29	30	70

Gene No.	cDNA Clone ID	ATCC Deposit No:Z and Date	Vector	NT SEQ ID NO: X	Total NT Seq.	5' NT of Clone Seq.	3' NT of Clone Seq.	5' NT of Start Codon	5' NT of First AA of Signal Pep	AA SEQ ID NO: Y	First AA of Sig Pep	Last AA of Sig Pep	First AA of Secreted Portion	Last AA of ORF
84	HKAEG43	209965 06/11/98	pCMVSPORT 2.0	233	1286	1	1286	21	21	522	1	29	30	70
85	HKDBF34	209511 12/03/97	pCMVSPORT 1	95	1432	60	1418	69	69	384	1	14	15	222
85	HKDBF34	209511 12/03/97	pCMVSPORT 1	234	1356	1	1356	18	18	523	1	19	20	104
86	HKISB57	209603 01/29/98	pBluescript	96	1492	1	1439	130	130	385	1	19	20	95
87	HKIYP40	209463 11/14/97	pBluescript	97	1215	1	1215	43	43	386	1	32	33	76
88	HKMLK53	209511 12/03/97	pBluescript	98	1543	1	1543	20	20	387	1	25	26	69
89	HLDON23	209628 02/12/98	pCMVSPORT 3.0	99	1262	208	1256	368	368	388	1	20	21	113
90	HLDOW79	PTA-1544 03/21/00	pCMVSPORT 3.0	100	989	1	989	43	43	389	1	21	22	275
91	HLJB61	PTA-848 10/13/99	pCMVSPORT 1	101	1191	1	1191	158	158	390	1	29	30	38
91	HLJB61	PTA-848 10/13/99	pCMVSPORT 1	235	628	1	628	227	227	524	1	29	30	38
92	HLQDH79	209551 12/12/97	Lambda ZAP II	102	913	1	913	205	205	391	1	19	20	58
93	HLTHG37	209965 06/11/98	Uni-ZAP XR	103	3740	1908	3740	50	50	392	1	1	2	319
93	HLTHG37	209965 06/11/98	Uni-ZAP XR	236	1932	98	1932	313	313	525	1	35	36	42

Gene No.	cDNA Clone ID	ATCC Deposit No:Z and Date	Vector	NT SEQ ID NO: X	Total NT Seq.	5' NT of Clone Seq.	3' NT of Clone Seq.	5' NT of Start Codon	5' NT of First AA of Signal Pep	AA SEQ ID NO: Y	First AA of Sig Pep	Last AA of Sig Pep	First AA of Secreted Portion	Last AA of ORF
94	HLWAE11	203071 07/27/98	pCMVSPORT 3.0	104	1618	1	1618	28	28	393	1	46	47	278
95	HLWAY54	209651 03/04/98	pCMVSPORT 3.0	105	1892	1	1892	38	38	394	1	25	26	338
96	HLWCF05	209126 06/19/97	pCMVSPORT 3.0	106	646	1	646	155	155	395	1	36	37	58
97	HLYAF80	209126 06/19/97	pSPORT1	107	826	1	826	222	222	396	1	24	25	47
98	HLYAZ61	209022 05/08/97	pSPORT1	108	1237	1	1237	190	190	397	1	18	19	222
98	HLYAZ61	209022 05/08/97	pSPORT1	237	997	74	997	205	205	526	1	18	19	215
99	HMADS41	209563 12/18/97	Uni-ZAP XR	109	1267	1	1267	267	267	398	1	21	22	88
100	HMADU73	209139 07/03/97	Uni-ZAP XR	110	3194	1	3194	491	491	399	1	16	17	713
100	HMADU73	209139 07/03/97	Uni-ZAP XR	238	437	1	437	115	115	527	1	15	16	77
101	HMIAL37	209563 12/18/97	Uni-ZAP XR	111	1420	1	1420	49	49	400	1	13	14	97
102	HMKCG09	209346 10/09/97	pSPORT1	112	921	60	921	221	221	401	1	28	29	49
103	HMMAH60	209368 10/16/97	pSPORT1	113	822	1	822	142	142	402	1	15	16	50
104	HMQDT36	209022 05/08/97	Uni-ZAP XR	114	1871	1	1871	157	157	403	1	32	33	406

Gene No.	cDNA Clone ID	ATCC Deposit No:Z and Date	Vector	NT SEQ ID NO: X	Total NT Seq.	5' NT of Clone Seq.	3' NT of Clone Seq.	5' NT of Start Codon	5' NT of First AA of Signal Pep	AA SEQ ID NO: Y	First AA of Sig Pep	Last AA of Sig Pep	First AA of Secreted Portion	Last AA of ORF
104	HMQDT36	209022 05/08/97	Uni-ZAP XR	239	1914	37	1897	192	192	528	1	32	33	406
105	HMSHS36	PTA-2070 06/09/00	Uni-ZAP XR	115	1402	1	1402	134	134	404	1	23	24	103
105	HMSHS36	PTA-2070 06/09/00	Uni-ZAP XR	240	616	30	616	162	162	529	1	23	24	103
106	HMSJU68	209076 05/22/97	Uni-ZAP XR	116	1123	4	1123	272	272	405	1	31	32	49
107	HMSKC04	203105 08/13/98	Uni-ZAP XR	117	1417	1	1417	133	133	406	1	22	23	73
108	HMTAD67	209551 12/12/97	pCMV Sport 3.0	118	1173	1	1173	306	306	407	1	19	20	84
109	HMWFO02	209324 10/02/97	Uni-ZAP XR	119	547	1	547	7	7	408	1	37	38	68
109	HMWFO02	209324 10/02/97	Uni-ZAP XR	241	708	1	708	20	20	530	1	38	39	60
110	HMWFY10	209147 07/17/97	Uni-ZAP XR	120	556	1	556	367	367	409	1	15	16	30
110	HMWFY10	209147 07/17/97	Uni-ZAP XR	242	556	1	556		129	531	1	9	10	18
111	HNGBT31	97976 04/04/97	Uni-ZAP XR	121	639	1	639	224	224	410	1	28	29	104
112	HNGEO29	209299 09/25/97	Uni-ZAP XR	122	491	1	491	98	98	411	1	32	33	44
113	HNGIQ46	209243 09/12/97	Uni-ZAP XR	123	527	1	527	221	221	412	1	21	22	70

Gene No.	cDNA Clone ID	ATCC Deposit No:Z and Date	Vector	NT SEQ ID NO: X	Total NT Seq.	5' NT of Clone Seq.	3' NT of Clone Seq.	5' NT of Start Codon	5' NT of First AA of Signal Pep	AA SEQ ID NO: Y	First AA of Sig Pep	Last AA of Sig Pep	First AA of Secreted Portion	Last AA of ORF
114	HNGJP69	209603 01/29/98	Uni-ZAP XR	124	985	1	985	321	321	413	1	14	15	74
115	HNGOI12	PTA-847 10/13/99	Uni-ZAP XR	125	2128	1	2128	27	27	414	1	34	35	57
115	HNGOI12	PTA-847 10/13/99	Uni-ZAP XR	243	774	1	774	27	27	532	1	34	35	57
115	HNGOI12	PTA-847 10/13/99	Uni-ZAP XR	244	1396	1	1396		596	533	1	25	26	93
116	HNHOD46	PTA-1543 03/21/00	Uni-ZAP XR	126	1355	1	1355	12	12	415	1	20	21	80
117	HNTBL27	209324 10/02/97	pCMVSPORT 3.0	127	791	71	791	100	100	416	1	23	24	115
118	HNTNI01	209782 04/20/98	pSport1	128	2087	1	2087	307	307	417	1	33	34	76
118	HNTNI01	209782 04/20/98	pSport1	245	1274	1	1114	306	306	534	1	33	34	49
119	HOAAC90	209236 09/04/97	Uni-ZAP XR	129	642	1	642	33	33	418	1	15	16	104
119	HOAAC90	209236 09/04/97	Uni-ZAP XR	246	652	1	652	38	38	535	1	15	16	104
120	HOCNF19	203570 01/11/99	pSport1	130	1118	1	1118	166	166	419	1	20	21	87
121	HODDW40	209463 11/14/97	Uni-ZAP XR	131	682	1	682	139	139	420	1	19	20	40
122	HODFN71	203570 01/11/99	Uni-ZAP XR	132	1126	1	1126		1	421	1	1	2	159

Gene No.	cDNA Clone ID	ATCC Deposit No:Z and Date	Vector	NT SEQ ID NO: X	Total NT Seq.	5' NT of Clone Seq.	3' NT of Clone Seq.	5' NT of Start Codon	5' NT of First AA of Signal Pep	AA SEQ ID NO: Y	First AA of Sig Pep	Last AA of Sig Pep	First AA of Secreted Portion	Last AA of ORF
122	HODFN71	203570 01/11/99	Uni-ZAP XR	247	1124	1	1124	27	27	536	1	18	19	148
123	HOEBZ89	203517 12/10/98	Uni-ZAP XR	133	2520	1	2520	19	19	422	1	21	22	333
124	HOEDB32	209628 02/12/98	Uni-ZAP XR	134	1462	73	1462	104	104	423	1	21	22	226
125	HOEDH84	209965 06/11/98	Uni-ZAP XR	135	2079	1	2079	256	256	424	1	20	21	404
126	HOFNC14	PTA-623 09/02/99	pCMVSPORT 2.0	136	2794	1	2794	79	79	425	1	13	14	73
126	HOFNC14	PTA-623 09/02/99	pCMVSPORT 2.0	248	3095	1	3095	155	155	537	1	13	14	72
127	HOFND85	PTA-1544 03/21/00	pCMVSPORT 2.0	137	2048	1	2048	167	167	426	1	22	23	627
128	HOFOC33	PTA-848 10/13/99	pCMVSPORT 2.0	138	1669	1	1669	76	76	427	1	21	22	363
128	HOFOC33	PTA-848 10/13/99	pCMVSPORT 2.0	249	518	1	518	81	81	538	1	21	22	112
128	HOFOC33	PTA-848 10/13/99	pCMVSPORT 2.0	250	518	1	518	81	81	539	1	17	18	112
128	HOFOC33	PTA-848 10/13/99	pCMVSPORT 2.0	251	1670	1	1670	76	76	540	1	21	22	139
128	HOFOC33	PTA-848 10/13/99	pCMVSPORT 2.0	252	606	1	606		23	541	1			7
128	HOFOC33	PTA-848 10/13/99	pCMVSPORT 2.0	253	841	1	841		158	542	1	6	7	14

Gene No.	cDNA Clone ID	ATCC Deposit No:Z and Date	Vector	NT SEQ ID NO: X	Total NT Seq.	5' NT of Clone Seq.	3' NT of Clone Seq.	5' NT of Start Codon	5' NT of First AA of Signal Pep	AA SEQ ID NO: Y	First AA of Sig Pep	Last AA of Sig Pep	First AA of Secreted Portion	Last AA of ORF
128	HOFOC33	PTA-848 10/13/99	pCMVSPORT 2.0	254	868	1	847		3	543	1	1	2	288
129	HOGCK20	209853 05/07/98	pCMVSPORT 2.0	139	2087	1	2087	57	57	428	1	23	24	522
129	HOGCK20	209853 05/07/98	pCMVSPORT 2.0	255	2075	1	2054		53	544	1	22	23	554
130	HOGCS52	PTA-848 10/13/99	pCMVSPORT 2.0	140	2571	1	2571	25	25	429	1	22	23	453
130	HOGCS52	PTA-848 10/13/99	pCMVSPORT 2.0	256	2645	1	2586	30	30	545	1	22	23	453
130	HOGCS52	PTA-848 10/13/99	pCMVSPORT 2.0	257	1098	457	638		2	546	1	1	2	96
131	HOUCQ17	209086 05/29/97	Uni-ZAP XR	141	4712	1	4693	508	508	430	1	51	52	967
132	HOUDK26	209423 10/30/97	Uni-ZAP XR	142	1051	1	1051	214	214	431	1	30	31	174
133	HOVCA92	209299 09/25/97	pSPORT	143	707	1	488	181	181	432	1	20	21	62
134	HPJBK12	PTA-855 10/18/99	Uni-ZAP XR	144	2648	1	2648	126	126	433	1	18	19	48
134	HPJBK12	PTA-855 10/18/99	Uni-ZAP XR	258	538	1	538	119	119	547	1	18	19	48
134	HPJBK12	PTA-855 10/18/99	Uni-ZAP XR	259	1346	1	1346		969	548	1			10
134	HPJBK12	PTA-855 10/18/99	Uni-ZAP XR	260	912	1	912	509	509	549	1			4

Gene No.	cDNA Clone ID	ATCC Deposit No:Z and Date	Vector	NT SEQ ID NO: X	Total NT Seq.	5' NT of Clone Seq.	3' NT of Clone Seq.	5' NT of Start Codon	5' NT of First AA of Signal Pep	AA SEQ ID NO: Y	First AA of Sig Pep	Last AA of Sig Pep	First AA of Secreted Portion	Last AA of ORF
135	HPJEX20	PTA-872 10/26/99	Uni-ZAP XR	145	566	1	566	23	23	434	1	26	27	174
135	HPJEX20	PTA-872 10/26/99	Uni-ZAP XR	261	1823	1	1823	31	31	550	1	23	24	115
135	HPJEX20	PTA-872 10/26/99	Uni-ZAP XR	262	1964	1	1964	170	170	551	1	23	24	174
135	HPJEX20	PTA-872 10/26/99	Uni-ZAP XR	263	769	1	769	84	84	552	1	23	24	228
135	HPJEX20	PTA-872 10/26/99	Uni-ZAP XR	264	818	1	818		565	553	1	1	2	84
136	HPMAI22	209683 03/20/98	Uni-ZAP XR	146	1274	334	1274	483	483	435	1	16	17	59
137	HPRBC80	209852 05/07/98	Uni-ZAP XR	147	2543	1245	2543	94	94	436	1	30	31	387
137	HPRBC80	209852 05/07/98	Uni-ZAP XR	265	2052	275	2032	404	404	554	1	26	27	69
138	HPRSB76	209244 09/12/97	pBluescript	148	741	1	741	127	127	437	1	22	23	59
139	HPWAY46	PTA-843 10/13/99	Uni-ZAP XR	149	1414	1	1414	468	468	438	1	30	31	52
139	HPWAY46	PTA-843 10/13/99	Uni-ZAP XR	266	891	1	891	474	474	555	1	30	31	52
139	HPWAY46	PTA-843 10/13/99	Uni-ZAP XR	267	501	120	501		178	556	1	1	2	86

Gene No.	cDNA Clone ID	ATCC Deposit No:Z and Date	Vector	NT SEQ ID NO: X	Total NT Seq.	5' NT of Clone Seq.	3' NT of Clone Seq.	5' NT of Start Codon	5' NT of AA of Signal Pep	AA SEQ ID NO: Y	First AA of Sig Pep	Last AA of Sig Pep	First AA of Secreted Portion	Last AA of ORF
140	HPWAZ95	209007 04/28/97 209083 05/29/97	Uni-ZAP XR	150	323	1	323	88	88	439	1	27	28	78
141	HRACD15	209852 05/07/98	pCMVSPORT 3.0	151	1539	24	1539	252	252	440	1	40	41	53
141	HRACD15	209852 05/07/98	pCMVSPORT 3.0	268	1681	24	1453	252	252	557	1	40	41	53
142	HRDFD27	209423 10/30/97	Uni-ZAP XR	152	805	1	805	82	82	441	1	35	36	83
143	HSAVD46	209124 06/19/97	Uni-ZAP XR	153	773	2	767	155	155	442	1	20	21	58
144	HSAWZ41	209463 11/14/97	Uni-ZAP XR	154	1388	1	1388	98	98	443	1	24	25	57
145	HSAYM40	209139 07/03/97	Uni-ZAP XR	155	433	1	433	190	190	444	1	19	20	63
146	HSDEZ20	209852 05/07/98	Uni-ZAP XR	156	795	1	795	58	58	445	1	41	42	122
146	HSDEZ20	209852 05/07/98	Uni-ZAP XR	269	1540	1	1540	66	66	558	1	41	42	97
147	HSDJA15	203081 07/30/98	Uni-ZAP XR	157	1443	1	1443	247	247	446	1	20	21	152
148	HSDSB09	209145 07/17/97	pBluescript	158	809	1	809	16	16	447	1	17	18	135
148	HSDSB09	209145 07/17/97	pBluescript	270	819	1	819	22	22	559	1	17	18	121

Gene No.	cDNA Clone ID	ATCC Deposit No:Z and Date	Vector	NT SEQ ID NO: X	Total NT Seq.	5' NT of Clone Seq.	3' NT of Clone Seq.	5' NT of Start Codon	5' NT of AA of Signal Pep	AA SEQ ID NO: Y	First AA of Sig Pep	Last AA of Sig Pep	First AA of Secreted Portion	Last AA of ORF
149	HSFAM31	209346 10/09/97	Uni-ZAP XR	159	868	1	868	44	44	448	1			9
150	HSXAX21	209853 05/07/98	Uni-ZAP XR	160	1986	1	1986	177	177	449	1	13	14	72
151	HSQCM10	209641 02/25/98	Uni-ZAP XR	161	657	1	654	130	130	450	1	19	20	62
152	HSSGD52	PTA-1543 03/21/00	Uni-ZAP XR	162	2425	1	2425	344	344	451	1	32	33	606
152	HSSGD52	PTA-1543 03/21/00	Uni-ZAP XR	271	2460	105	2460	338	338	560	1	27	28	606
153	HSSJC35	209853 05/07/98	Uni-ZAP XR	163	1174	1	1174	62	62	452	1	28	29	295
153	HSSJC35	209853 05/07/98	Uni-ZAP XR	272	1163	1	1163	55	55	561	1	30	31	295
153	HSSJC35	209853 05/07/98	Uni-ZAP XR	273	1183	1	1183	66	66	562	1	30	31	37
154	HSXEC75	209641 02/25/98	Uni-ZAP XR	164	1112	1	1112	295	295	453	1	33	34	45
155	HSYAZ50	PTA-849 10/13/99	pCMVSPORT 3.0	165	1097	1	1097	131	131	454	1	18	19	56
155	HSYAZ50	PTA-849 10/13/99	pCMVSPORT 3.0	274	768	226	768	345	345	563	1	18	19	56
155	HSYAZ50	PTA-849 10/13/99	pCMVSPORT 3.0	275	2087	770	875		723	564	1	1	2	106
155	HSYAZ50	PTA-849 10/13/99	pCMVSPORT 3.0	276	2096	1767	2050		2	565	1	1	2	279

Gene No.	cDNA Clone ID	ATCC Deposit No:Z and Date	Vector	NT SEQ ID NO: X	Total NT Seq.	5' NT of Clone Seq.	3' NT of Clone Seq.	5' NT of Start Codon	5' NT of First AA of Signal Pep	AA SEQ ID NO: Y	First AA of Sig Pep	Last AA of Sig Pep	First AA of Secreted Portion	Last AA of ORF
156	HSYBG37	209463 11/14/97	pCMVSPORT 3.0	166	1238	1	1238	47	47	455	1	24	25	305
156	HSYBG37	209463 11/14/97	pCMVSPORT 3.0	277	1239	1	1239	48	48	566	1	24	25	305
157	HSZAF47	209124 06/19/97	Uni-ZAP XR	167	1304	1	1304	106	106	456	1	16	17	289
157	HSZAF47	209124 06/19/97	Uni-ZAP XR	278	1333	2	1333	107	107	567	1	18	19	127
158	HTADX17	209124 06/19/97	Uni-ZAP XR	168	1147	0	1148	92	92	457	1	23	24	142
158	HTADX17	209124 06/19/97	Uni-ZAP XR	279	1140	22	1140	84	84	568	1	19	20	142
159	HTEAF65	PTA-322 07/09/99	Uni-ZAP XR	169	563	1	563	135	135	458	1	19	20	75
160	HTEBI28	209177 07/24/97	Uni-ZAP XR	170	413	1	413	43	43	459	1	20	21	67
161	HTEHR24	209224 08/28/97	Uni-ZAP XR	171	1075	50	1075	84	84	460	1	29	30	163
161	HTEHR24	209224 08/28/97	Uni-ZAP XR	280	1038	1	1038	41	41	569	1	28	29	124
162	HTEHU31	209568 01/06/98	Uni-ZAP XR	172	1113	1	1113	121	121	461	1	25	26	312
163	HTEHU93	209090 06/05/97	Uni-ZAP XR	173	738	1	738	188	188	462	1	24	25	142
163	HTEHU93	209090 06/05/97	Uni-ZAP XR	281	745	1	745	187	187	570	1	24	25	113

Gene No.	cDNA Clone ID	ATCC Deposit No:Z and Date	Vector	NT SEQ ID NO: X	Total NT Seq.	5' NT of Clone Seq.	3' NT of Clone Seq.	5' NT of Start Codon	5' NT of First AA of Signal Pep	AA SEQ ID NO: Y	First AA of Sig Pep	Last AA of Sig Pep	First AA of Secreted Portion	Last AA of ORF
164	HTEIP36	209244 09/12/97	Uni-ZAP XR	174	752	1	752	22	22	463	1	19	20	58
165	HTEIV80	209511 12/03/97	Uni-ZAP XR	175	1748	1	1748	203	203	464	1	14	15	47
166	HTEPG70	203570 01/11/99	Uni-ZAP XR	176	813	1	813	365	365	465	1	27	28	89
167	HTHBG43	PTA-843 10/13/99	Uni-ZAP XR	177	848	1	848	47	47	466	1			39
167	HTHBG43	PTA-843 10/13/99	Uni-ZAP XR	282	632	103	632	149	149	571	1			39
168	HTJMA95	209853 05/07/98	pCMVSPORT 2.0	178	1650	198	1569	527	527	467	1	22	23	181
169	HTLFE42	209138 07/03/97	Uni-ZAP XR	179	712	1	712	116	116	468	1	22	23	77
170	HTLIT32	203570 01/11/99	Uni-ZAP XR	180	1074	164	897	288	288	469	1	26	27	246
171	HTLIV19	PTA-2081 06/09/00	Uni-ZAP XR	181	978	1	978	110	110	470	1	33	34	84
172	HTOAK16	209368 10/16/97	Uni-ZAP XR	182	1466	1	1466	87	87	471	1	18	19	110
173	HTOHD42	203081 07/30/98	Uni-ZAP XR	183	946	1	946	155	155	472	1	24	25	190
174	HTOHEM15	PTA-843 10/13/99	Uni-ZAP XR	184	1949	1	1949	30	30	473	1	20	21	61
174	HTOHEM15	PTA-843 10/13/99	Uni-ZAP XR	283	408	1	408	23	23	572	1	20	21	61

Gene No.	cDNA Clone ID	ATCC Deposit No.:Z and Date	Vector	NT SEQ ID NO: X	Total NT Seq.	5' NT of Clone Seq.	3' NT of Clone Seq.	5' NT of Start Codon	5' NT of First AA of Signal Pep	AA SEQ ID NO: Y	First AA of Sig Pep	Last AA of Sig Pep	First AA of Secreted Portion	Last AA of ORF
174	HTOHM15	PTA-843 10/13/99	Uni-ZAP XR	284	1299	982	1274		71	573	1	1	2	322
174	HTOHM15	PTA-843 10/13/99	Uni-ZAP XR	285	1669	1	1622		1555	574	1	9	10	13
175	HTPBW79	209511 12/03/97	Uni-ZAP XR	185	1374	1	1374	178	178	474	1	22	23	362
175	HTPBW79	209511 12/03/97	Uni-ZAP XR	286	1515	118	1507	302	302	575	1	24	25	362
175	HTPBW79	209511 12/03/97	Uni-ZAP XR	287	1404	1	1404	92	92	576	1	22	23	415
176	HTTDB46	203484 11/17/98	Uni-ZAP XR	186	3059	1	3059	55	55	475	1	17	18	318
176	HTTDB46	203484 11/17/98	Uni-ZAP XR	288	2008	215	2008	153	153	577	1	17	18	461
177	HTXCV12	209423 10/30/97	Uni-ZAP XR	187	1134	1	1134	175	175	476	1	27	28	102
177	HTXCV12	209423 10/30/97	Uni-ZAP XR	289	1162	1	1162	183	183	578	1	27	28	91
178	HTXDW56	209746 04/07/98	Uni-ZAP XR	188	1583	1	1583	217	217	477	1	21	22	201
179	HTXFL30	209603 01/29/98	Uni-ZAP XR	189	1991	1	1991	30	30	478	1	39	40	102
180	HTXKP61	203364 10/19/98	Uni-ZAP XR	190	1209	1	1209	169	169	479	1	33	34	42
181	HUKAH51	209568 01/06/98	Lambda ZAP II	191	853	1	853	286	286	480	1	20	21	151

Gene No.	cDNA Clone ID	ATCC Deposit No.:Z and Date	Vector	NT SEQ ID NO: X	Total NT Seq.	5' NT of Clone Seq.	3' NT of Clone Seq.	5' NT of Start Codon	5' NT of First AA of Signal Pep	AA SEQ ID NO: Y	First AA of Sig Pep	Last AA of Sig Pep	First AA of Secreted Portion	Last AA of ORF
181	HUKAH51	209568 01/06/98	Lambda ZAP II	290	754	1	754	144	144	579	1	22	23	142
181	HUKAH51	209568 01/06/98	Lambda ZAP II	291	667	1	667	55	55	580	1	22	23	119
182	HUKBT29	209746 04/07/98	Lambda ZAP II	192	1757	56	1757	74	74	481	1	19	20	506
183	HUSBA88	PTA-623 09/02/99	Lambda ZAP II	193	2733	27	2733	270	270	482	1	15	16	615
184	HWBAR88	PTA-867 10/26/99	pCMV/Sport 3.0	194	1051	1	1051	156	156	483	1	18	19	75
185	HWBCB89	PTA-499 08/11/99	pCMV/Sport 3.0	195	1317	3	1317	37	37	484	1	19	20	187
185	HWBCB89	PTA-499 08/11/99	pCMV/Sport 3.0	292	1315	1	1315	35	35	581	1	19	20	187
186	HWBCP79	209641 02/25/98	pCMV/Sport 3.0	196	1138	1	1138	243	243	485	1	21	22	105
186	HWBCP79	209641 02/25/98	pCMV/Sport 3.0	293	1138	1	1138	233	233	582	1	21	22	105
187	HWHGP71	203858 03/18/99	pCMV/Sport 3.0	197	1021	1	1021	389	389	486	1	51	52	211
187	HWHGP71	203858 03/18/99	pCMV/Sport 3.0	294	1037	1	1037	394	394	583	1	18	19	77
188	HWHQ55	203027 06/26/98	pCMV/Sport 3.0	198	3282	1	3282	169	169	487	1	26	27	742
189	HWLEV32	PTA-884 10/28/99	pSport1	199	1218	1	1218	39	39	488	1	18	19	45

Gene No.	cDNA Clone ID	ATCC Deposit No:Z and Date	Vector	NT SEQ ID NO: X	Total NT Seq.	5' NT of Clone Seq.	3' NT of Clone Seq.	5' NT of Start Codon	5' NT of First AA of Signal Pep	AA SEQ ID NO: Y	First AA of Sig Pep	Last AA of Sig Pep	First AA of Secreted Portion	Last AA of ORF
189	HWLEV32	PTA-884 10/28/99	pSport1	295	1203	1	1203	29	29	584	1	18	19	45
189	HWLEV32	PTA-884 10/28/99	pSport1	296	1144	528	596		3	585	1	1	2	136
189	HWLEV32	PTA-884 10/28/99	pSport1	297	1120	791	851		1	586	1	1	2	141
190	HYBAR01	209580 01/14/98	Uni-ZAP XR	200	1440	1	1440	157	157	489	1	26	27	46
191	HYBBE75	203570 01/11/99	Uni-ZAP XR	201	838	1	838	319	319	490	1	25	26	41
192	HAPSA79	PTA-1543 03/21/00	Uni-ZAP XR	202	4386	1	4386	468	468	491	1	30	31	310
192	HAPSA79	PTA-1543 03/21/00	Uni-ZAP XR	298	4385	1	4385	468	468	587	1	30	31	310
192	HAPSA79	PTA-1543 03/21/00	Uni-ZAP XR	299	4386	1	4386	468	468	588	1	30	31	310
193	HDPJM30	209563 12/18/97	pCMVSPORT 3.0	203	1635	308	1633	59	59	497	1	59	60	525
194	HHFGR93	209746 04/07/98	Uni-ZAP XR	204	1835	1	1835	132	132	498	1	29	30	390
195	HJBVA55	203364 10/19/98	pBluescript SK-	205	2441	39	2429	238	238	499	1	26	27	58

Table 1B (Comprised of Tables 1B.1 and 1B.2)

The first column in Table 1B.1 and Table 1B.2 provides the gene number in the application corresponding to the clone identifier. The second column in Table 1B.1 and Table 1B.2 provides a unique "Clone ID:" for the cDNA clone related to each contig sequence disclosed in Table 1B.1 and Table 1B.2. This clone ID references the cDNA clone which contains at least the 5' most sequence of the assembled contig and at least a portion of SEQ ID NO:X as determined by directly sequencing the referenced clone. The referenced clone may have more sequence than described in the sequence listing or the clone may have less. In the vast majority of cases, however, the clone is believed to encode a full-length polypeptide. In the case where a clone is not full-length, a full-length cDNA can be obtained by methods described elsewhere herein. The third column in Table 1B.1 and Table 1B.2 provides a unique "Contig ID" identification for each contig sequence. The fourth column in Table 1B.1 and Table 1B.2 provides the "SEQ ID NO:" identifier for each of the contig polynucleotide sequences disclosed in Table 1B.

Table 1B.1

The fifth column in Table 1B.1, "ORF (From-To)", provides the location (i.e., nucleotide position numbers) within the polynucleotide sequence "SEQ ID NO:X" that delineate the preferred open reading frame (ORF) shown in the sequence listing and referenced in Table 1B.1, column 6, as SEQ ID NO:Y. Where the nucleotide position number "To" is lower than the nucleotide position number "From", the preferred ORF is the reverse complement of the referenced polynucleotide sequence. The sixth column in Table 1B.1 provides the corresponding SEQ ID NO:Y for the polypeptide sequence encoded by the preferred ORF delineated in column 5. In one embodiment, the invention provides an amino acid sequence comprising, or alternatively consisting of, a polypeptide encoded by the portion of SEQ ID NO:X delineated by "ORF (From-To)". Also provided are polynucleotides encoding such amino acid sequences and the complementary strand thereto. Column 7 in Table 1B.1 lists residues comprising epitopes contained in the polypeptides encoded by the preferred ORF (SEQ ID NO:Y), as predicted using the algorithm of Jameson and Wolf, (1988) *Comp. Appl. Biosci.* 4:181-186. The Jameson-Wolf antigenic analysis was performed using the computer program PROTEAN (Version 3.11 for the Power MacIntosh, DNASTAR, Inc., 1228 South Park Street Madison, WI). In specific embodiments, polypeptides of the invention comprise, or alternatively consist of, at least one, two, three, four, five or more of the predicted epitopes as described in Table 1B. It will be appreciated that depending on the analytical criteria used to predict antigenic determinants, the exact address of the determinant may vary slightly.

Column 8 in Table 1B.1 provides a chromosomal map location for certain polynucleotides of the invention. Chromosomal location was determined by finding exact matches to EST and cDNA sequences contained in the NCBI (National Center for Biotechnology

Information) UniGene database. Each sequence in the UniGene database is assigned to a “cluster”; all of the ESTs, cDNAs, and STSs in a cluster are believed to be derived from a single gene. Chromosomal mapping data is often available for one or more sequence(s) in a UniGene cluster; this data (if consistent) is then applied to the cluster as a whole. Thus, it is possible to infer the chromosomal location of a new polynucleotide sequence by determining its identity with a mapped UniGene cluster.

A modified version of the computer program BLASTN (Altshul, et al., J. Mol. Biol. 215:403-410 (1990), and Gish, and States, Nat. Genet. 3:266-272) (1993) was used to search the UniGene database for EST or cDNA sequences that contain exact or near-exact matches to a polynucleotide sequence of the invention (the ‘Query’). A sequence from the UniGene database (the ‘Subject’) was said to be an exact match if it contained a segment of 50 nucleotides in length such that 48 of those nucleotides were in the same order as found in the Query sequence. If all of the matches that met this criteria were in the same UniGene cluster, and mapping data was available for this cluster, it is indicated in Table 1B under the heading “Cytologic Band”. Where a cluster had been further localized to a distinct cytologic band, that band is disclosed; where no banding information was available, but the gene had been localized to a single chromosome, the chromosome is disclosed.

Once a presumptive chromosomal location was determined for a polynucleotide of the invention, an associated disease locus was identified by comparison with a database of diseases which have been experimentally associated with genetic loci. The database used was the Morbid Map, derived from OMIM™ and National Center for Biotechnology Information, National Library of Medicine (Bethesda, MD) 2000;. If the putative chromosomal location of a polynucleotide of the invention (Query sequence) was associated with a disease in the Morbid Map database, an OMIM reference identification number was noted in column 9, Table 1B.1, labelled “OMIM Disease Reference(s). Table 5 is a key to the OMIM reference identification numbers (column 1), and provides a description of the associated disease in Column 2.

Table 1B.2

Column 5, in Table 1B.2, provides an expression profile and library code:count for each of the contig sequences (SEQ ID NO:X) disclosed in Table 1B, which can routinely be combined with the information provided in Table 4 and used to determine the tissues, cells, and/or cell line libraries which predominantly express the polynucleotides of the invention. The first number in Table 1B.2, column 5 (preceding the colon), represents the tissue/cell source identifier code corresponding to the code and description provided in Table 4. The second number in column 5 (following the colon) represents the number of times a sequence corresponding to the reference polynucleotide sequence was identified in the corresponding tissue/cell source. Those tissue/cell source identifier codes in which the first two letters are “AR” designate

information generated using DNA array technology. Utilizing this technology, cDNAs were amplified by PCR and then transferred, in duplicate, onto the array. Gene expression was assayed through hybridization of first strand cDNA probes to the DNA array. cDNA probes were generated from total RNA extracted from a variety of different tissues and cell lines. Probe synthesis was performed in the presence of ^{33}P dCTP, using oligo (dT) to prime reverse transcription. After hybridization, high stringency washing conditions were employed to remove non-specific hybrids from the array. The remaining signal, emanating from each gene target, was measured using a Phosphorimager. Gene expression was reported as Phosphor Stimulating Luminescence (PSL) which reflects the level of phosphor signal generated from the probe hybridized to each of the gene targets represented on the array. A local background signal subtraction was performed before the total signal generated from each array was used to normalize gene expression between the different hybridizations. The value presented after "[array code]:" represents the mean of the duplicate values, following background subtraction and probe normalization. One of skill in the art could routinely use this information to identify normal and/or diseased tissue(s) which show a predominant expression pattern of the corresponding polynucleotide of the invention or to identify polynucleotides which show predominant and/or specific tissue and/or cell expression.

TABLE 1B.1

Gene No:	cDNA Clone ID	Contig ID:	SEQ ID NO: X	ORF (From-To)	AA SEQ ID NO: Y	Predicted Epitopes	Cytologic Band	OMIM Disease Reference(s):
1	H2MAC30	544957	11	157 - 375	300	Pro-54 to Gly-67.		
2	H6EDF66	520498	12	146 - 538	301			
3	H6EDX46	1352262	13	229 - 774	302	Arg-21 to Leu-26, Arg-88 to Asn-104, Arg-111 to Ser-116, Arg-154 to Lys-160, Cys-164 to Asp-169.	12q15	181430, 600698, 600698, 600698, 600698, 600808, 602116
	H6EDX46	637786	203	128 - 382	492	Arg-21 to Leu-26.		
4	HABAG37	637942	14	97 - 285	303	Thr-24 to Gly-42, Glu-53 to Gly-58.	19p13.3	108725, 120700, 133171, 136836, 145981, 147141, 164953, 188070, 600957, 601238, 601846, 602216, 602477
5	HACBD91	637482	15	117 - 266	304		3q13.33	600882
6	HACCI17	891114	16	461 - 1114	305	Ser-201 to Tyr-217.	22q11.21	123620, 151410, 600850
	HACCI17	731877	204	135 - 353	493			
7	HAGAM64	626997	17	57 - 191	306	Arg-30 to Tyr-39.		
8	HAHDR32	635357	18	435 - 980	307	Met-1 to Ser-7, Asp-41 to Met-48, Pro-61 to Ser-67, Pro-121 to Trp-130, His-161 to Lys-181.	3p14.3-p14.1	150250, 156845, 156845, 156845, 156845, 164500, 277730, 600971, 601226
9	HAIBO71	490848	19	325 - 525	308			
10	HAF57	823516	20	43 - 324	309	Cys-25 to Ile-31, Cys-85 to Asn-91.		
11	HAMFC93	904749	21	136 - 711	310	Asp-31 to Pro-36, Ser-88 to Gln-95, Ala-163 to Glu-171.	6q27	152200, 167000, 600320, 600883, 602544

	HAMFC93	900586	205	115 - 651	494	Asp-31 to Pro-36, Ser-88 to Gln-95.		
	HAMFC93	906819	206	323 - 349	495			
12	HAPNY86	587261	22	100 - 489	311	Pro-27 to Leu-41.		
13	HATDF29	845965	23	143 - 1300	312	Ser-35 to Ser-44, Ser-86 to Leu-91, Asp-143 to Leu-150, Lys-166 to Ser-171, Ser-208 to Gly-213, Lys-239 to Leu-244, Glu-317 to Asn-324.		
14	HBAFJ33	625916	24	60 - 392	313	Gln-66 to Cys-71, Thr-76 to Gly-81, His-87 to Asp-92.	14q32	123270, 245200, 251600, 270100, 276900
15	HBAFV19	843036	25	6 - 779	314	Pro-12 to Phe-18, Ser-139 to Pro-146, Asp-162 to Arg-173, Thr-188 to Glu-204, Lys-245 to Gly-258.		
16	HBIBW67	553678	26	685 - 798	315	Met-1 to Tyr-8.		
17	HBIMB51	963208	27	98 - 535	316	His-24 to Ala-29, Glu-42 to Glu-49, Arg-63 to Thr-80, Gln-100 to Lys-119, Lys-141 to Gln-146.		
	HBIMB51	672711	207	93 - 485	496	His-24 to Ala-29, Glu-42 to Glu-49.		
18	HBJID05	1130660	28	157 - 756	317	Lys-82 to Pro-87, Leu-110 to Lys-129.		
	HBJID05	544980	208	137 - 472	497	Lys-82 to Pro-90.		
19	HBJU28	561723	29	133 - 387	318	Gln-23 to Asn-31, Tyr-42 to Ser-58.		

20	HBJLH40	828130	30	74 - 298	319	Ile-69 to Pro-74.			
21	HBXFL29	842802	31	560 - 733	320	Arg-36 to Pro-43.	17q22-q23	106180, 109270, 109270, 109270, 109270, 109270, 120150, 120150, 120150, 138700, 139250, 148065, 148080, 150200, 154275, 171190, 176960, 185800, 221820, 249000, 253250, 600525, 600852, 601844	
22	HCACU58	625923	32	137 - 388	321				
23	HCDBW86	520435	33	139 - 231	322				
24	HCE3G69	728432	34	165 - 1175	323	Lys-50 to Asp-66, Pro-68 to Glu-77, Glu-102 to Glu-107, Glu-131 to Leu-146, Ala-175 to Glu-183, Phe-205 to Lys-216, Val-263 to Thr-281, Pro-304 to Ala-313.	2q36.1	120070, 120131, 120131, 138030, 259900	
	HCE3G69	494346	209	165 - 482	498	Lys-50 to Leu-69.			
25	HCEEA88	634967	35	134 - 316	324	Asn-28 to Pro-34.	21q22.2	176261, 601399	
26	HCEFB69	748245	36	188 - 862	325	Gln-189 to Gly-195.			
27	HCFMV71	526599	37	31 - 207	326	Arg-35 to Gly-44.			
28	HCNSD93	630649	38	139 - 279	327				
29	HCUIM65	550208	39	557 - 700	328				
30	HCWKC15	553621	40	37 - 159	329	Lys-28 to Thr-34.			
31	HCWLD74	628256	41	138 - 335	330				
32	HDHEB60	499233	42	568 - 894	331	Asp-48 to Ser-54.	11p11.2	133701, 168500, 171650, 176930, 176930, 600623, 600811, 600958	
33	HDHMA45	902513	43	199 - 1440	332	Ala-145 to Ser-154, Ala-258 to Tyr-263, Ala-287 to Arg-297, Thr-306 to Met-316.	11q		
	HDHMA45	812764	210	204 - 1445	499	Ala-145 to Ser-154, Ala-258 to Tyr-263,			

34	HDHMA72	547772	44	287 - 1234	333	Ala-287 to Arg-297, Thr-306 to Met-316. Glu-67 to Asn-74, Glu-88 to Asn-93, Lys-95 to Ser-105, Arg-152 to Ala-164, Ala-204 to Arg-210, Phe-254 to Thr-262, Pro-295 to His-311.	7q36	142335, 152427, 163729, 176450, 190605, 600510, 600725
35	HDPBA28	1062783	45	259 - 3084	334	Gln-33 to Trp-49, Gly-161 to Gly-172, Ile-207 to Arg-212, Asn-414 to Val-419, Val-423 to Gln-428, Val-436 to Gly-441, Lys-467 to Leu-478, Phe-497 to Ser-508, Met-550 to Gly-560, Glu-688 to Thr-697, Ile-711 to Gly-720, Ala-747 to Gly-759, Leu-785 to Phe-791, Ser-795 to Gln-800, Thr-808 to Lys-813, Ser-821 to Phe-832, Thr-879 to Glu-889, Leu-898 to Gln-904, Gln-934 to Met-941. Gln-33 to Trp-49, Gly-161 to Gly-172, Ile-207 to Arg-212, Asn-414 to Val-419, Val-423 to Gln-428,	5q14.3	
	HDPBA28	866429	211	69 - 2894	500			

							Val-436 to Gly-441, Lys-467 to Leu-478, Phe-497 to Ser-508, Met-550 to Gly-560, Glu-688 to Thr-697, Ile-711 to Gly-720, Ala-747 to Gly-759, Leu-785 to Phe-791, Ser-795 to Gln-800.			
36	HDP CO25	460682	46	182 - 343	335		Pro-22 to His-33, Ser-42 to Trp-48.			
37	HDP CY37	837699	47	76 - 1809	336		Pro-23 to His-34, Thr-64 to Trp-71.	12q13.3		181430, 232800, 600808, 601284, 601769, 601769, 602116
	HDP CY37	604114	212	76 - 870	501		Pro-23 to His-34, Thr-64 to Trp-71, Lys-245 to Ala-252.			
38	HDP HI51	460679	48	245 - 367	337		Gly-2 to Glu-7, Arg-27 to Gly-34.			
39	HDP ND46	637586	49	15 - 1469	338		Ala-107 to Ser-112.			
40	HDP OH06	683371	50	252 - 980	339		Met-1 to Ser-8.			
41	HDP SP54	744440	51	2356 - 2499	340		Pro-29 to Lys-37.	1q21.2		104770, 107670, 110700, 145001, 146760, 146790, 191315, 601412, 601652, 601863, 602491
	HDP SP54	502472	213	179 - 343	502					
42	HDP VH60	796865	52	8 - 163	341			16q13		114835, 132700, 172490, 600968
43	HDP WN93	992925	53	45 - 2453	342		Pro-36 to Ser-52, Ala-63 to Pro-78, Ala-106 to Lys-115, Glu-134 to Glu-141, Val-155 to Asp-164, Phe-199 to Gly-204, Arg-218 to Leu-228,	17q21.33		109270, 109270, 109270, 109270, 109270, 120150, 120150, 120150, 148065, 148080, 154275, 171190, 185800, 221820, 249000, 253250, 600119, 600119, 600525, 601844

								Glu-230 to Val-235, Val-247 to Pro-253, Arg-262 to Gly-276, Thr-303 to Gln-310, Arg-335 to Trp-342, Glu-399 to Ala-415, Ser-458 to Glu-466, Arg-508 to Asp-517, Glu-580 to Pro-585, Gln-620 to Trp-628, Lys-651 to Ala-657, Gly-677 to Met-682, Ala-712 to Leu-717, Gly-724 to Thr-731, Arg-770 to Gln-775.			
	HDPWN93	887914	214	35 - 679	503			Pro-36 to Ser-52, Ala-63 to Pro-78, Ala-106 to Lys-115, Glu-134 to Glu-141, Val-155 to Asp-164.			
	HDPWN93	905983	215	27 - 158	504						
44	HDQHD03	1309175	54	274 - 1266	343			Arg-26 to Lys-46, Ala-70 to Lys-81, Gln-100 to Pro-105, Val-118 to Leu-123, Pro-166 to Pro-171, Gly-310 to Gly-331.			
	HDQHD03	834692	216	259 - 1257	505			Arg-26 to Lys-46, Ala-70 to Lys-81, Phe-92 to Gly-98.			
45	HE2EN04	545008	55	57 - 209	344						
46	HE8QV67	1050076	56	502 - 744	345				9		
	HE8QV67	1050077	217	256 - 1500	506			Gln-29 to Lys-35,			

							Lys-48 to Gln-54, Arg-80 to Asp-90, Pro-166 to Arg-173, Glu-178 to Tyr-188, Glu-220 to Leu-228, Ile-246 to Pro-253, Arg-281 to Asp-288, Ser-305 to His-313, Asn-319 to Asp-328, Asp-361 to Phe-366, Arg-372 to Tyr-377, Gly-384 to Ser-402.			
47	HE8UB86	834913	57	201 - 953	346	Pro-43 to Cys-52, Lys-105 to Ser-113.				
48	HE9BK23	675382	58	39 - 968	347	Arg-18 to Asp-27, Leu-29 to Arg-36, Ser-90 to Tyr-104, Val-108 to Lys-114.	1p31.1-p22.3	600309, 601414, 602094		
49	HEBBN36	486120	59	645 - 806	348		17q21.31	109270, 109270, 109270, 109270, 109270, 120150, 120150, 120150, 148065, 148080, 154275, 171190, 185800, 221820, 249000, 253250, 600119, 600119, 600525, 601844		
50	HEQCC55	1352368	60	25 - 411	349	Pro-35 to Trp-42, Ala-53 to Asp-62, Arg-103 to Phe-110, Ile-114 to Glu-120.	16p13.3	141750, 141800, 141800, 141800, 141800, 141850, 141850, 141850, 141850, 141850, 156850, 186580, 191092, 600140, 600273, 601313, 601785		
	HEQCC55	884824	218	62 - 397	507	Pro-35 to Trp-42, Pro-65 to Asp-72, Thr-86 to Phe-93, Ile-97 to Glu-103.				
	HEQCC55	748227	219	57 - 524	508	Pro-35 to Trp-42, Pro-65 to Asp-72, Thr-86 to Glu-92,				

							Pro-96 to Gly-104, Ser-138 to Gly-154.			
51	HESAJ10	526013	61	405 - 620	350					
52	HETEU28	1018676	62	256 - 717	351		Glu-80 to Trp-85, Gly-91 to Asp-99, Leu-106 to Leu-116, Trp-120 to Pro-146.			
	HETEU28	882328	220	331 - 792	509		Glu-80 to Trp-85, Gly-91 to Pro-97.			
53	HFABG18	847073	63	53 - 316	352		Glu-36 to Lys-55.	19q13	109560, 205900, 600652, 600757	
54	HFAMB72	490697	64	559 - 741	353		Gln-53 to Thr-60.			
55	HFCCQ50	579993	65	47 - 1105	354		Ala-27 to Ser-38, Pro-43 to Asn-54, Thr-115 to Asp-121, Leu-225 to Val-232, Pro-247 to Gly-252, Arg-306 to Leu-311.	12q24	113100, 124200, 147440, 158590, 160781, 163950, 163950, 251170, 276710, 600175, 601517	
56	HFIIIZ70	1043350	66	24 - 167	355			22		
	HFIIIZ70	906708	221	74 - 217	510					
57	HFKET18	889515	67	137 - 361	356		Lys-60 to Ser-74.			
58	HFLNB64	580829	68	62 - 199	357			8p23-p22	148370, 238600, 238600, 238600, 238600, 600143, 601385, 602629	
59	HFOXA73	850699	69	25 - 180	358			12,12p13		
	HFOXA73	532079	222	15 - 68	511					
60	HFPAC12	589522	70	140 - 406	359		Thr-26 to Glu-33.	5q33.2	109690, 109690, 164770, 180071	
61	HFP AO71	629193	71	414 - 809	360		Pro-43 to Pro-50, Asn-65 to Gly-70.			
62	HFPCX36	526635	72	103 - 243	361					
63	HFPCX64	1309796	73	181 - 444	362		Lys-60 to Asn-67.			
	HFPCX64	877637	223	181 - 723	512		Lys-60 to Asn-67.			
	HFPCX64	638851	224	257 - 520	513		Lys-60 to Asn-67.			
	HFPCX64	514187	225	257 - 517	514					

64	HFTBM50	545012	74	158 - 262	363	Ala-19 to Lys-34.	4q12	103600, 103600, 103600, 104150, 104150, 104500, 164920, 164920, 164920, 170650, 600900
65	HFXDJ75	626114	75	44 - 169	364	Pro-31 to Pro-37.		
66	HFXJU68	1352218	76	141 - 626	365		1p33	120260, 138140, 178300, 246450
	HFXJU68	570855	226	148 - 348	515			
67	HGBIB74	837220	77	14 - 1144	366	Ser-67 to Glu-74, Arg-81 to Val-86, Tyr-147 to Asp-160.	20q11.21	
	HGBIB74	838602	227	28 - 540	516	Ser-67 to Glu-74, Arg-81 to Val-86, Tyr-147 to Asp-160.		
	HGBIB74	899864	228	2 - 454	517	Ser-3 to Gln-10, Val-14 to Gln-19, Asp-32 to His-40, Gly-50 to His-55, Pro-76 to Ser-87.		
68	HHEMA75	494099	78	569 - 823	367	Lys-74 to Tyr-79.	7q33	180105, 222800
69	HHENK42	493724	79	63 - 191	368		7	
70	HHEPM33	877639	80	269 - 517	369	Met-1 to Thr-13, Ser-27 to Phe-34, Arg-53 to Pro-59, Ser-77 to Ser-82.	2q36.1	120070, 120131, 120131, 138030, 259900
71	HHEPT60	463027	81	245 - 355	370		19p13.3	108725, 120700, 133171, 136836, 145981, 147141, 164953, 188070, 600957, 601238, 601846, 602216, 602477
72	HHFHJ59	411332	82	192 - 530	371	Pro-32 to Ser-39.		
73	HHGDW43	554613	83	107 - 241	372	Ser-39 to Ser-44.		
74	HHPEC09	695726	84	71 - 238	373	Tyr-39 to Arg-51.		
75	HHPTJ65	490904	85	247 - 393	374			
76	HJABX32	487807	86	557 - 712	375	Trp-29 to Gly-42, Gly-46 to His-51.		

77	HJBCU04	877643	87	96 - 626	376	Met-1 to Cys-7, Gln-45 to Gly-61, Gln-77 to Thr-93, Arg-113 to Arg-118, Ser-135 to Glu-147, Gln-155 to Ala-161.	9p13-p12	230400, 250250	
78	HJMBN89	565675	88	348 - 518	377		14q32.33	144120, 147020, 147110	
79	HJMBW30	491209	89	110 - 238	378	Pro-30 to Ala-35.			
80	HKABI84	565078	90	274 - 417	379	Phe-25 to Ser-30./	1p32-p34	120950, 120960, 130500, 133200, 138140, 168360, 171760, 171760, 176100, 176100, 178300, 187040, 230000, 255800, 600101, 600650, 600650, 600722, 600722	
81	HKACB56	554616	91	27 - 269	380	Tyr-39 to Lys-58.			
82	HKACM93	1352383	92	218 - 2293	381	Ser-5 to Trp-10, Ala-30 to Glu-39, Arg-66 to Trp-72, Glu-84 to Arg-97, Glu-159 to Gly-176, Ile-189 to Glu-197, Glu-206 to Arg-215, Arg-218 to Gly-227, Gly-316 to Ala-322, Pro-430 to Val-435, Pro-446 to Gly-452, Ser-488 to Gly-504, Glu-569 to Lys-575, Pro-581 to Cys-588, Ala-687 to Glu-692.	1		
	HKACM93	907084	229	189 - 548	518	Ser-5 to Trp-10, Ala-30 to Glu-39, Arg-66 to Trp-72, Glu-84 to Arg-97.			
	HKACM93	907085	230	314 - 1120	519	Ser-5 to Trp-10,			

							Ala-30 to Glu-39, Arg-66 to Trp-72, Glu-84 to Arg-97, Glu-159 to Gly-176, Ile-189 to Glu-197, Glu-206 to Arg-215, Arg-218 to His-226.			
	HKACM93	906154	231	202 - 255	520		Trp-2 to Met-16.			
	HKACM93	906150	232	638 - 775	521		Gln-24 to Gly-31, Pro-33 to Ala-38.			
83	HKADQ91	604123	93	229 - 1056	382		Cys-31 to Arg-36, Asp-81 to His-86, Asn-264 to Met-275.			
84	HKAEG43	889521	94	32 - 241	383		Pro-41 to Cys-47, Phe-52 to Gly-59, Pro-62 to His-70.			
	HKAEG43	753273	233	21 - 233	522		Pro-41 to Cys-47, Phe-52 to Gly-59, Pro-62 to His-70.			
85	HKDBF34	833065	95	69 - 734	384		Lys-60 to Ala-66, Arg-169 to Cys-186, Asp-199 to Gly-205, Thr-214 to Leu-219.	Xp22	300000, 300066, 300077, 300310, 301220, 302350, 304050, 304110, 306100, 309530, 309585, 312040	
	HKDBF34	587268	234	18 - 332	523		Lys-60 to Ala-66, Thr-78 to Ser-83.			
86	HKISB57	625956	96	130 - 417	385		Ala-23 to Arg-36, His-38 to Ala-46, Pro-50 to Gly-56, Arg-85 to Val-94.	22q12.2	101000, 101000, 101000, 101000, 123620, 138981, 188826, 600850, 601669	
87	HKIYP40	580845	97	43 - 273	386		Ala-66 to Leu-73.			
88	HKMLK53	587269	98	20 - 229	387		Gly-27 to Cys-35.	2q35	118800, 123660, 125660, 125660, 193500, 193500, 193500, 193500, 201460, 205100,	

89	HLDON23	636083	99	368 - 709	388	Arg-28 to Gln-36.	15q23	237300, 262000, 600266, 601277 118485, 151670, 231680, 272800, 272800, 272800, 276700, 600374, 601780
90	HLDOW79	847396	100	43 - 870	389	Pro-171 to Gln-179, Leu-218 to Lys-225, Phe-266 to Cys-275.		
91	HLJB161	1019012	101	158 - 274	390		19	
	HLJB161	833665	235	227 - 343	524			
92	HLQDH79	588446	102	205 - 381	391		3p21.2-p21.3	116806, 120120, 120120, 120120, 120436, 120436, 120436, 138320, 168468, 182280, 238310, 600163, 601226
93	HLTHG37	787530	103	50 - 1006	392	Asn-36 to Lys-42, Lys-53 to Gln-60, Ile-64 to Ala-77, Ala-128 to Tyr-135, Lys-184 to Ala-199, Leu-245 to Leu-250.		
	HLTHG37	743169	236	313 - 441	525			
94	HLWAE11	783071	104	28 - 861	393	Asp-55 to Asp-67, Ser-76 to His-81, Lys-96 to Gly-103, Met-111 to Gly-133, Gln-222 to Ile-228, Lys-250 to Tyr-258.	22q13.1	103050, 103050, 124030, 124030, 138981, 182380, 188826, 190040, 190040, 190040
95	HLWAY54	658702	105	38 - 1054	394	Asp-27 to Ser-32, Pro-52 to Thr-58, Arg-63 to Asn-70, Gln-78 to Gly-83, Thr-107 to Asn-113, Thr-160 to Val-176, Ser-188 to Gly-241, Leu-248 to Pro-265,	12p13.31	125370, 601458

							Asp-91 to Gln-100, Glu-122 to Ser-128, Arg-137 to Pro-143, Asp-157 to Asn-162, Glu-168 to Asn-174, Ser-199 to Gly-206, Pro-213 to Ala-218, Glu-251 to Thr-257, Ser-353 to His-361, Gly-363 to Ala-375, Pro-382 to Phe-387, Arg-401 to Leu-406.			
	HMQDT36	424085	239	192 - 1412	528		Glu-78 to Asn-83, Asp-91 to Gln-100, Glu-122 to Ser-128, Arg-137 to Pro-143, Asp-157 to Asn-162, Glu-168 to Asn-174, Ser-199 to Gly-206, Pro-213 to Ala-218, Glu-251 to Thr-257, Ser-353 to His-361, Gly-363 to Ala-375, Pro-382 to Phe-387, Arg-401 to Leu-406.			
105	HMSHS36	1127691	115	134 - 445	404		Thr-28 to Arg-49, Ser-57 to Arg-64, Pro-72 to His-78.			
	HMSHS36	1028961	240	162 - 473	529		Thr-28 to Arg-49, Ser-57 to Arg-64.			
106	HMSJU68	427121	116	272 - 421	405		Met-1 to Gly-7.			
107	HMSKC04	799540	117	133 - 354	406		Thr-27 to Arg-33, Gly-37 to Ser-42,			

108	HMTAD67	588447	118	306 - 560	407	Pro-52 to Arg-72. Pro-43 to Leu-49, Pro-61 to Gly-66, Ser-71 to Ser-83. Pro-60 to Arg-68.			
109	HMWFO02	1352198	119	7 - 210	408				
	HMWFO02	542061	241	20 - 202	530				
110	HMWFO10	825421	120	367 - 456	409				
	HMWFO10	490495	242	129 - 185	531				
111	HNGBT31	408334	121	224 - 538	410	Ala-83 to Thr-91.			
112	HNGEO29	532622	122	98 - 232	411	Met-1 to Arg-8, Leu-35 to Glu-41.			
113	HNGIQ46	526651	123	221 - 433	412	Ala-28 to Gly-34, Pro-57 to Thr-66.			
114	HNGJP69	604891	124	321 - 545	413				
115	HNGOI12	1041375	125	27 - 200	414	Met-1 to Gly-9.	11		
	HNGOI12	838184	243	27 - 200	532	Met-1 to Gly-9.			
	HNGOI12	839283	244	596 - 877	533				
116	HNHOD46	843488	126	12 - 251	415				
117	HNTBL27	545534	127	100 - 447	416	Arg-45 to Thr-52, Tyr-60 to Gly-66, Ala-87 to Trp-92, Leu-105 to Ser-115. Lys-71 to Trp-76.	3p21.31	116806, 168468, 182280, 212138, 600163	
118	HNTNI01	1352285	128	307 - 534	417				
	HNTNI01	699848	245	306 - 455	534				
119	HOAAC90	1301202	129	33 - 347	418	Trp-25 to Pro-33, Gln-88 to Pro-93.			
	HOAAC90	518979	246	38 - 352	535	Trp-25 to Pro-33, Gln-88 to Pro-93.			
120	HOCNF19	835049	130	166 - 429	419	Thr-45 to Pro-56, Ser-66 to Lys-74.			
121	HODDW40	579256	131	139 - 261	420				
122	HODFN71	1194866	132	1 - 477	421	Lys-50 to Phe-57,			

							Ser-70 to Arg-77, Tyr-81 to Ser-87, Pro-112 to Thr-117.			
	HODFN71	834999	247	27 - 473	536		Lys-39 to Phe-46, Ser-59 to Arg-66, Tyr-70 to Ser-76, Pro-101 to Thr-106.			
123	HOEBZ89	828177	133	19 - 1020	422		Lys-34 to Glu-39, Ile-47 to Ser-53, Pro-106 to Leu-111, Pro-140 to Gly-146, Glu-195 to Gly-204, Leu-281 to Thr-288, Glu-291 to Arg-297, Tyr-302 to Ile-308.			
124	HOEDB32	634994	134	104 - 784	423		Pro-34 to Ser-43, Glu-54 to Ser-60.	17q11.2		154275, 162200, 162200, 182138, 239100, 600881, 601954, 602403
125	HOEDH84	748236	135	256 - 1467	424		Ser-74 to Ala-84, Gln-156 to Tyr-161, Tyr-184 to Asn-189, Ser-218 to Ile-223, Pro-299 to Ser-308, His-359 to Thr-368, Tyr-390 to Asp-404.			
126	HOFNC14	1352378	136	79 - 297	425					
	HOFNC14	899292	248	155 - 373	537					
127	HOFND85	847424	137	167 - 2047	426		Asp-216 to Gly-224, Asp-268 to Asn-274, Thr-285 to Lys-290, Asp-339 to Pro-345, Ile-356 to Pro-361, Arg-371 to Asn-378, Ala-408 to Tyr-417,			

128	HOFOC33	1186156	138	76 - 1167	427	Pro-429 to Gln-434, Arg-461 to Pro-466, Ala-475 to Ala-482.			
						Thr-28 to Tyr-40, Gln-61 to Ser-68, Glu-74 to Lys-95, Glu-163 to Thr-169, Arg-197 to His-204, Ser-210 to Phe-216, Thr-272 to Asp-278, Arg-286 to Gly-291, Cys-310 to Ala-316.			
	HOFOC33	967554	249	81 - 419	538	Thr-28 to Tyr-40, Gln-61 to Ser-68, Glu-74 to Leu-94.			
	HOFOC33	878690	250	81 - 419	539	Thr-28 to Tyr-40, Gln-61 to Ser-68.			
	HOFOC33	905734	251	76 - 495	540	Thr-28 to Tyr-40, Gln-61 to Ser-68, Glu-74 to Lys-95, Thr-119 to Leu-124, Pro-126 to Gln-131.			
	HOFOC33	902326	252	23 - 46	541				
	HOFOC33	885140	253	158 - 202	542				
	HOFOC33	806819	254	3 - 866	543				
129	HOGCK20	745445	139	57 - 1622	428	Pro-25 to Arg-31, Thr-52 to Val-63, Asn-129 to Lys-135, Gln-197 to Trp-202, Thr-230 to Glu-236, Pro-242 to Tyr-248, Leu-280 to Pro-291,	20q12-q13.12	600281, 600281, 602025	

							Ser-348 to Ser-356, Pro-362 to Gln-368, Thr-398 to His-406, Trp-430 to Leu-435, Glu-499 to Gly-504.			
	HOGCK20	664499	255	53 - 1717	544		Pro-24 to Arg-30, Thr-51 to Val-62, Asn-128 to Lys-134, Gln-196 to Trp-201, Thr-229 to Glu-235, Pro-241 to Tyr-247, Leu-279 to Pro-290, Ser-347 to Ser-355, Pro-361 to Gln-367, Thr-397 to His-405, Trp-429 to Leu-434, Gln-510 to Val-518.			
130	HOGCS52	919898	140	25 - 1383	429		Met-28 to Arg-34, Thr-154 to Arg-173, Gly-180 to Tyr-185, Leu-226 to Asp-231, Leu-272 to Lys-277, Thr-378 to Asn-383, Asp-421 to Tyr-433, Leu-442 to Ala-451.			
	HOGCS52	907118	256	30 - 1391	545		Met-28 to Arg-34, Thr-154 to Arg-173, Gly-180 to Tyr-185, Leu-226 to Asp-231, Leu-272 to Lys-277, Thr-378 to Asn-383, Asp-421 to Arg-431.			
	HOGCS52	867965	257	2 - 289	546		Ala-1 to Ala-6.			

131	HOUQC17	429229	141	508 - 3408	430	Gly-8 to Leu-14, Met-18 to Phe-30.		
132	HOUDK26	565393	142	214 - 735	431	Ser-139 to Ser-144, Phe-153 to Leu-159, Gln-162 to Ser-170.		
133	HOVCA92	527644	143	181 - 369	432			
134	HPJBK12	1011467	144	126 - 272	433		4,8	
	HPJBK12	525375	258	119 - 265	547			
	HPJBK12	796925	259	969 - 1001	548			
	HPJBK12	699587	260	509 - 523	549			
135	HPJEX20	1352420	145	23 - 544	434	Gln-102 to Ser-108.	1	
	HPJEX20	1184442	261	31 - 375	550			
	HPJEX20	975252	262	170 - 694	551	Gln-102 to Ser-108.		
	HPJEX20	894744	263	84 - 767	552			
	HPJEX20	898220	264	565 - 816	553	Ser-23 to Thr-32, Ala-37 to Gln-44.		
136	HPMAI22	635491	146	483 - 662	435			
137	HPRBC80	829136	147	94 - 1254	436	Asp-6 to His-13, Asp-114 to Gly-131, Thr-166 to Gln-181, Val-210 to Thr-216, Pro-222 to Tyr-227.	2p21	120435, 120435, 126600, 135300, 136435, 152790, 152790, 157170, 182601, 601771
	HPRBC80	720095	265	404 - 613	554			
138	HPRSB76	526310	148	127 - 306	437		15q11-q13	103581, 146150, 176270, 218000, 227220, 601623, 601800, 601889, 602117
139	HPWAY46	1001560	149	468 - 626	438		4	
	HPWAY46	876469	266	474 - 632	555			
	HPWAY46	789574	267	178 - 435	556			
140	HPWAZ95	413270	150	88 - 321	439			
141	HRACD15	871221	151	252 - 410	440			
	HRACD15	706332	268	252 - 413	557			
142	HRDFD27	567004	152	82 - 333	441			

143	HSAVD46	456536	153	155 - 328	442				
144	HSAWZ41	580872	154	98 - 271	443				
145	HSAYM40	462797	155	190 - 381	444				
146	HSDEZ20	1352287	156	58 - 423	445				
	HSDEZ20	704101	269	66 - 359	558				
147	HSDJA15	795252	157	247 - 705	446				
148	HSDSB09	1301498	158	16 - 423	447				
	HSDSB09	463645	270	22 - 387	559				
149	HSFAM31	552789	159	44 - 73	448				
150	HSHAX21	612823	160	177 - 392	449				
151	HSQCM10	638591	161	130 - 318	450				
152	HSSGD52	1352343	162	344 - 2161	451				
	HSSGD52	845666	271	338 - 2155	560				

156	HSYBG37	1056317	166	47 - 964	455	Lys-153 to Lys-163, Trp-245 to Ala-251, Phe-259 to Gly-273. Ser-47 to Pro-57, Ser-77 to Glu-82, Thr-90 to Trp-98, Arg-124 to Lys-137, Ala-183 to Glu-192, Lys-220 to Gln-229, Asn-244 to Arg-258, Thr-271 to Asn-278, Glu-285 to Gly-297.	16p13.3	141750, 141800, 141800, 141800, 141800, 141850, 141850, 141850, 141850, 141850, 156850, 186580, 191092, 600140, 600273, 601313, 601785	
	HSYBG37	581098	277	48 - 965	566	Ser-47 to Pro-57, Ser-77 to Glu-82, Thr-90 to Trp-98, Arg-124 to Lys-137, Ala-183 to Glu-192, Lys-220 to Gln-229, Asn-244 to Arg-258, Thr-271 to Asn-278, Glu-285 to Gly-297.			
157	HSZAF47	1352172	167	106 - 972	456	Gly-16 to Pro-30, Pro-42 to Gly-56, Gly-62 to Gly-77, Glu-93 to Gly-104, Glu-109 to Glu-114, Pro-121 to Gly-134, Ser-157 to Arg-162, Glu-174 to Thr-182, Ile-283 to Leu-289.	4p16-p15	225500, 600593, 602363	
	HSZAF47	456551	278	107 - 490	567	Gly-16 to Pro-30, Pro-42 to Gly-56, Gly-62 to Gly-77,			

168	HTJMA95	706618	178	527 - 1069	467	Gly-85 to Lys-94, Gln-125 to Cys-131, Glu-151 to Gly-159.	15q25	231680, 276700
169	HTLFE42	460583	179	116 - 349	468	Ser-22 to Thr-32, Pro-37 to Ser-42.		
170	HTLIT32	833906	180	288 - 1028	469	Ser-83 to Tyr-88, Ala-129 to Ser-134, Ser-227 to Ala-233.		
171	HTLIV19	1046341	181	110 - 364	470		3	
172	HTOAK16	560744	182	87 - 419	471	Asp-27 to Ser-36.		
173	HTOHD42	604983	183	155 - 727	472	Gly-33 to Arg-40, Ser-106 to Met-112, Ala-154 to Gly-163.		
174	HTOHM15	1028538	184	30 - 215	473			
	HTOHM15	848199	283	23 - 208	572			
	HTOHM15	848200	284	71 - 1036	573	Arg-1 to Gly-7, Phe-11 to Arg-23.		
	HTOHM15	848196	285	1555 - 1596	574			
175	HTPBW79	1317835	185	178 - 1263	474	Leu-21 to Ala-30, Ser-38 to Asp-47, Pro-87 to Asp-94, Leu-197 to Thr-204, Pro-256 to Ser-262, Thr-277 to Arg-282, Thr-293 to Val-302, Lys-315 to Arg-321.	11	
	HTPBW79	581435	286	302 - 1390	575	Leu-21 to Ala-30, Ser-38 to Asp-47, Pro-87 to Asp-94, Leu-197 to Thr-204, Pro-256 to Ser-262, Thr-277 to Arg-282,		

							Pro-115 to Arg-127, Pro-140 to Gln-151.			
	HUKAH51	1300737	290	144 - 572	579		Trp-35 to Trp-45, Pro-52 to Asp-57, Thr-73 to Thr-80, Pro-96 to Leu-103, Pro-106 to Arg-118, Pro-131 to Gln-142.			
	HUKAH51	603538	291	55 - 414	580		Trp-35 to Trp-45, Pro-52 to Asp-57, Thr-73 to Thr-80, Pro-96 to Leu-103, Pro-106 to Leu-119.			
182	HUKBT29	694590	192	74 - 1594	481		Thr-35 to Lys-43, Pro-59 to Arg-64.	1q42		106150, 106150, 145260, 173870, 173870, 600759, 600996, 601744, 601975
183	HUSBA88	895435	193	270 - 2117	482		Glu-32 to Arg-38, Gln-56 to Asn-64, Ser-69 to His-83, Arg-87 to Gln-118, Leu-137 to Thr-146, Pro-148 to Gly-157, Trp-177 to Ala-184, Asp-188 to Ser-194, Lys-221 to Arg-227, Arg-283 to Pro-289, Pro-302 to Asp-308, Thr-328 to Phe-333, Ser-348 to Gly-353, Gly-392 to Leu-400, Arg-416 to Lys-422, Tyr-493 to Glu-502, Thr-527 to Trp-535, Asn-559 to Met-572.	9q34		125270, 125270, 128100, 137350, 191100, 215700, 223360, 268900, 601850

184	HWBAR88	836469	194	156 - 383	483			600320	
185	HWBCB89	1093347	195	37 - 600	484	Gln-20 to Phe-25, Gly-58 to Ala-66, Gln-69 to Leu-74, Asn-87 to Ile-100, Thr-135 to Trp-142.	1q24-q41	107300, 131210, 136132, 145001, 145260, 173610, 276901, 600332, 600759, 601518, 601652, 601744, 601975	
	HWBCB89	886210	292	35 - 598	581	Gln-20 to Phe-25, Gly-58 to Ala-66, Gln-69 to Leu-74, Asn-87 to Ile-100, Thr-135 to Trp-142.			
186	HWBCP79	846382	196	243 - 560	485	Trp-47 to Thr-54, Ser-68 to Asn-73, Ser-86 to Gly-92.			
	HWBCP79	646977	293	233 - 550	582	Trp-47 to Thr-54.			
187	HWHGP71	995431	197	389 - 1021	486	His-56 to Val-62, Gly-105 to His-113, Cys-141 to Trp-147, His-149 to Arg-155, Glu-159 to Pro-172.			
	HWHGP71	839250	294	394 - 627	583	Pro-49 to Ser-54, Thr-68 to Thr-77.			
188	HWHQ55	762842	198	169 - 2397	487	Val-35 to Lys-41, Ser-68 to Gln-73, Glu-88 to Glu-93, Arg-156 to Gly-163, Ala-199 to Gly-206, Asp-216 to Ser-226, Thr-249 to Asn-254, Asp-339 to Pro-345, Ile-370 to Gly-379, Pro-429 to Glu-434, Arg-461 to Pro-466,			

								Ala-475 to Thr-482, Pro-585 to Gly-593, Glu-631 to Gln-639, Pro-674 to Pro-682, Gln-715 to Gly-720, Ser-736 to Arg-742.			
189	HWLEV32	1032602	199	39 - 176	488						
	HWLEV32	873296	295	29 - 166	584						
	HWLEV32	881710	296	3 - 410	585						
	HWLEV32	846351	297	1 - 423	586			His-7 to Gly-15, Pro-89 to Arg-95, Pro-103 to His-109.			
190	HYBAR01	610383	200	157 - 297	489						
191	HYBBE75	834784	201	319 - 444	490			Pro-34 to Trp-41.			
192	HAPSA79	846517	202	468 - 1400	491			Leu-3 to Arg-8, Asp-57 to Arg-64, Glu-66 to Thr-75, Arg-120 to Ile-126, Gln-161 to Asp-177, Thr-182 to Ser-194, Lys-211 to Gln-216, Asn-274 to Gly-290, Thr-296 to Phe-302.			
	HAPSA79	887467	298	468 - 1400	587			Leu-3 to Arg-8, Asp-57 to Arg-64, Glu-66 to Thr-75, Arg-120 to Ile-126, Gln-161 to Asp-177, Thr-182 to Ser-194, Lys-211 to Gln-216, Asn-274 to Gly-290, Thr-296 to Phe-302.			

	HAPSA79	878627	299	468 - 1400	588	Leu-3 to Arg-8, Asp-57 to Arg-64, Glu-66 to Thr-75, Arg-120 to Ile-126, Gln-161 to Asp-177, Thr-182 to Ser-194, Lys-211 to Gln-216, Asn-274 to Gly-290, Thr-296 to Phe-302.		
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Table 1B.2

Gene No:	cDNA Clone ID	Contig ID:	SEQ ID NO:X	Tissue Distribution Library Code:Count (see Table 4 for Library Codes)				
1	H2MAC30	544957	11	AR096:11, AR039:10, AR313:10, AR299:10, AR250:9, AR240:8, AR254:8, AR055:8, AR242:8, AR060:7, AR089:7, AR162:7, AR316:6, AR161:6, AR163:6, AR213:6, AR269:6, AR252:5, AR268:5, AR169:5, AR200:5, AR204:5, AR215:5, AR165:5, AR053:5, AR196:5, AR166:5, AR164:5, AR199:5, AR104:5, AR282:5, AR176:5, AR266:5, AR180:4, AR264:4, AR261:4, AR277:4, AR300:4, AR229:4, AR183:4, AR181:4, AR190:4, AR173:4, AR263:4, AR247:4, AR309:4, AR197:4, AR274:4, AR178:4, AR214:4, AR205:4, AR212:4, AR243:4, AR312:4, AR191:4, AR253:4, AR182:4, AR236:4, AR170:4, AR245:3, AR185:3, AR272:3, AR217:3, AR171:3, AR267:3, AR175:3, AR308:3, AR192:3, AR290:3, AR271:3, AR193:3, AR291:3, AR219:3, AR237:3, AR233:3, AR188:3, AR201:3, AR216:3, AR311:3, AR270:3, AR177:3, AR174:3, AR218:3, AR234:3, AR283:3, AR179:3, AR293:3, AR207:3, AR231:3, AR221:3, AR228:3, AR203:3, AR285:3, AR262:3, AR255:2, AR224:2, AR288:2, AR238:2, AR195:2, AR287:2, AR257:2, AR239:2, AR168:2, AR286:2, AR189:2, AR296:2, AR230:2, AR223:2, AR275:2, AR289:2, AR297:1, AR222:1, AR232:1, AR033:1, AR260:1, AR061:1, AR227:1, AR295:1, AR235:1, AR294:1, AR225:1, AR258:1, AR172:1, AR226:1, AR210:1, AR211:1 L0766:16, L0743:11, H0692:8, L0769:7, L0518:6, L0748:6, L0771:4, L0745:4, L0779:4, H0265:3, S0358:3, H0494:3, L0755:3, L3814:2, H0550:2, H0486:2, H0581:2, H0135:2, L0761:2, L0804:2, L0774:2, L0438:2, L0777:2, H0685:1, S0114:1, H0583:1, S0116:1, S0212:1, H0254:1, S0408:1, S0476:1, H0772:1, T0104:1, H0586:1, H0587:1, H0331:1, T0109:1, H0599:1, L0738:1, H0150:1, H0012:1, H0264:1, S0438:1, L0770:1, L0374:1, L0764:1, L0768:1, L0803:1, L0653:1, L0776:1, L0788:1, L0792:1, L0663:1, S0428:1, S0053:1, S0216:1, H0783:1, L3811:1, S0152:1, H0522:1, H0555:1, S0432:1, L0744:1, L0751:1, L0749:1, L0756:1, L0758:1, S0436:1, L0601:1, H0543:1, H0423:1, S0424:1 and H0506:1.				
2	H6EDF66	520498	12	AR176:12, AR161:12, AR162:12, AR163:12, AR266:11, AR238:10, AR165:10, AR235:10, AR164:10, AR166:10, AR232:9, AR284:9, AR267:9, AR201:9, AR226:9, AR191:9, AR291:9, AR269:9, AR184:9, AR268:9, AR242:9,				

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3	H6EDX46	1352262	13	AR283:18, AR104:17, AR219:16, AR218:15, AR060:13, AR055:13, AR282:13, AR277:12, AR089:11, AR316:10, AR185:10, AR313:9, AR096:9, AR299:9, AR300:9, AR240:8, AR039:6, H0657:3, L0794:3, H0559:2, L0800:2, L0375:2, L0659:2, L0809:2, L0755:2, L0731:2, L0599:2, H0170:1, H0656:1, S0116:1, H0306:1, S0358:1, S0376:1, H0722:1, H0443:1, H0497:1, H0485:1, T0109:1, H0575:1, H0251:1, S0312:1, S0314:1, H0252:1, H0264:1, H0488:1, H0413:1, T0041:1, H0560:1, H0647:1, H0646:1, S0422:1, L0763:1, L0770:1, L0769:1, L0638:1, L0764:1, L0363:1, L0804:1, L0655:1, L0542:1, L0792:1, L0666:1, L0665:1, L3811:1, H0547:1, H0690:1, H0682:1, H0658:1, H0670:1, H0648:1, H0696:1, S0392:1, L0439:1, S0436:1, L0605:1, L0591:1, S0276:1, H0543:1 and H0423:1.
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4	HABAG37	637942	14	AR313:22, AR161:16, AR162:16, AR165:16, AR163:16, AR166:16, AR173:16, AR164:16, AR178:15, AR196:14, AR089:14, AR212:14, AR235:14, AR229:13, AR293:13, AR258:13, AR299:13, AR096:13, AR199:12, AR247:12, AR261:12, AR234:12, AR275:12, AR264:12, AR207:12, AR300:12, AR226:12, AR285:12, AR183:12, AR053:12, AR177:12, AR175:12, AR294:11, AR295:11, AR240:11, AR233:11, AR203:11, AR174:11, AR262:11, AR236:11, AR060:11, AR242:11, AR312:11, AR192:10, AR257:10, AR218:10, AR180:10, AR263:10, AR195:10, AR198:10, AR296:10, AR185:10, AR200:10, AR297:10, AR191:10, AR238:10, AR227:10, AR239:10, AR316:10, AR309:9, AR228:9, AR181:9, AR286:9, AR308:9, AR179:9, AR182:9, AR288:9, AR213:9, AR055:9, AR287:9, AR189:8, AR224:8, AR282:8, AR231:8, AR168:8, AR270:8, AR311:8, AR252:8, AR104:8, AR193:8, AR188:8, AR039:8, AR274:8, AR219:8, AR215:8, AR225:7, AR204:7, AR176:7, AR223:7, AR033:7, AR197:7, AR230:7, AR245:7, AR255:7, AR268:7, AR260:7, AR269:7, AR214:7, AR267:7, AR277:7, AR232:7, AR230:7, AR271:6, AR272:6, AR171:6, AR190:6, AR205:6, AR172:6, AR254:6, AR283:6, AR237:6, AR210:6, AR253:5, AR291:5, AR216:5, AR201:5, AR290:5, AR211:5, AR246:5, AR217:5, AR222:5, AR289:5, AR221:5, AR266:4, AR256:4, AR170:4, AR061:4, AR243:4, AR169:4, H0521:4, L0803:3, H0556:2, S0142:2, L0761:2, L0741:2, L0777:2, S0444:1, L0717:1, S0278:1, H0333:1, H0635:1, H0253:1, H0505:1, H0434:1, H0078:1, S0344:1, S0426:1, L0769:1, L5575:1, L0800:1, L0768:1, L0794:1, L0774:1, L0509:1, L0789:1, L0663:1, L0740:1, L0754:1, L0779:1, L0731:1 and L0759:1.
5	HACBD91	637482	15	AR055:116, AR283:103, AR060:91, AR089:55, AR235:53, AR299:52, AR185:51, AR104:49, AR096:34, AR039:30,

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7	HAGAM64	626997	17	AR169:7, AR170:4, AR171:3, AR168:2, AR180:2, AR183:2, AR188:2, AR257:2, AR311:1, AR264:1, AR178:1, AR261:1, AR313:1, AR308:1, AR243:1, AR225:1, AR235:1, AR196:1, AR282:1, AR255:1, AR096:1, AR269:1, AR277:1, S0030:1, S0010:1 and L0369:1.
8	HAHDR32	635357	18	AR055:27, AR235:20, AR283:12, AR236:10, AR266:9, AR261:9, AR161:9, AR162:9, AR163:8, AR176:8, AR293:8, AR256:7, AR197:6, AR089:6, AR295:6, AR180:6, AR294:6, AR262:6, AR204:6, AR289:6, AR165:6, AR182:6, AR296:6, AR285:6, AR164:5, AR166:5, AR060:5, AR291:5, AR181:5, AR239:5, AR250:5, AR297:5, AR268:5, AR257:5, AR229:5, AR267:5, AR309:5, AR183:5, AR260:5, AR228:5, AR237:5, AR272:5, AR178:5, AR201:4, AR214:4, AR193:4, AR299:4, AR053:4, AR253:4, AR223:4, AR271:4, AR316:4, AR269:4, AR287:4,

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11	HAMFC93	904749	21	AR254:4, AR171:3, AR207:3, AR170:3, AR169:3, AR053:3, AR213:2, AR225:2, AR271:2, AR165:2, AR198:2, AR201:2, AR166:2, AR176:2, AR264:2, AR282:2, AR272:2, AR089:2, AR297:2, AR288:2, AR257:2, AR188:2, AR224:2, AR175:1, AR163:1, AR283:1, AR196:1, AR162:1, AR246:1, AR308:1, AR226:1, AR161:1, AR193:1, AR164:1, AR183:1, AR285:1, AR173:1, AR286:1, AR255:1 H0561:1 AR316:210, AR089:205, AR313:203, AR205:188, AR096:145, AR299:140, AR219:119, AR245:111, AR185:109, AR252:108, AR218:107, AR039:106, AR274:101, AR246:96, AR055:91, AR195:89, AR283:88, AR272:88, AR271:83, AR212:83, AR214:80, AR254:77, AR053:77, AR197:75, AR213:73, AR204:72, AR216:72, AR201:72, AR243:68, AR282:67, AR222:66, AR312:66, AR242:63, AR060:62, AR277:62, AR165:62, AR308:61, AR193:60, AR198:60, AR309:59, AR164:59, AR300:59, AR104:57, AR311:57, AR223:57, AR162:56, AR161:56, AR250:54, AR166:54, AR163:54, AR192:54, AR217:53, AR253:51, AR264:50, AR263:49, AR169:49, AR240:44, AR224:41, AR247:39, AR275:39, AR171:35, AR168:34, AR179:31, AR207:31, AR174:30, AR172:30, AR225:30, AR210:28, AR236:27, AR177:26, AR189:26, AR291:26, AR178:25, AR180:24, AR288:24, AR211:24, AR183:23, AR296:22, AR230:22, AR061:22, AR033:22, AR173:22, AR175:21, AR170:21, AR295:21, AR297:21, AR268:21, AR269:21, AR293:21, AR266:21, AR290:20, AR270:20, AR181:20, AR267:20, AR237:20, AR231:20, AR289:19, AR229:17, AR285:17, AR238:17, AR176:17, AR215:16, AR221:16, AR234:16, AR190:15, AR226:15, AR233:15, AR182:14, AR286:14, AR235:14, AR239:14, AR287:14, AR232:14, AR227:13, AR294:13, AR255:13, AR256:13, AR228:13, AR199:12, AR261:11, AR257:10, AR258:10, AR262:10, AR188:9, AR260:8, AR200:6, AR203:6, AR191:5, AR196:4 L0439:20,

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13	HATDF29	845965	23	AR290:4, AR221:3, AR170:3, AR253:3, AR169:3, AR213:3, AR178:3, AR243:3, AR215:3, AR162:2, AR180:2, AR270:2, AR177:2, AR053:2, AR289:2, AR309:2, AR171:2, AR240:2, AR224:2, AR176:2, AR232:2, AR277:2, AR181:1, AR161:1, AR163:1, AR237:1, AR264:1, AR282:1, AR217:1, AR291:1, AR272:1, AR172:1, AR268:1, AR104:1, AR296:1, AR096:1, AR257:1, AR283:1 S0132:3, H0575:3, H0271:3, H0038:3, S0142:3, H0521:3, L0758:3, L0596:3, S0344:2, H0661:1, H0402:1, S0360:1, H0393:1, H0618:1, H0253:1, H0581:1, H0179:1, T0023:1, T0041:1, H0494:1, S0002:1, L0763:1, L0775:1, S0374:1, H0555:1, L0751:1 and L0589:1.
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22	HCACU58	625923	32	AR170:4, AR225:4, AR197:3, AR253:3, AR183:3, AR242:3, AR270:2, AR311:2, AR266:2, AR275:2, AR168:2, AR172:2, AR223:2, AR282:2, AR291:2, AR169:2, AR272:2, AR195:2, AR198:1, AR096:1, AR240:1, AR269:1, AR283:1, AR192:1, AR164:1, AR300:1, AR224:1, AR252:1 H0341:1, H0125:1, H0580:1, L0747:1 and L0749:1.
23	HCDBW86	520435	33	AR239:9, AR231:9, AR161:7, AR162:7, AR163:7, AR226:6, AR176:6, AR266:6, AR269:6, AR181:5, AR228:5, AR238:5, AR183:5, AR268:5, AR236:5, AR233:5, AR237:5, AR261:5, AR257:5, AR180:5, AR173:5, AR177:4, AR196:4, AR262:4, AR267:4, AR214:4, AR175:4, AR309:4, AR229:4, AR215:4, AR270:4, AR263:4, AR234:4, AR182:4, AR235:4, AR275:4, AR227:4, AR178:4, AR165:4, AR291:4, AR171:4, AR179:4, AR164:3, AR191:3, AR166:3, AR230:3, AR289:3, AR313:3, AR240:3, AR247:3, AR255:3, AR061:3, AR272:3, AR199:3, AR168:3, AR300:3, AR174:3, AR170:3, AR222:3, AR293:3, AR296:3, AR258:3, AR288:3, AR190:2, AR188:2, AR286:2, AR290:2, AR264:2, AR207:2, AR053:2, AR294:2, AR297:2, AR224:2, AR287:2, AR285:2, AR200:2, AR312:2, AR277:2, AR282:2, AR225:2, AR295:2, AR203:2, AR189:2, AR172:2, AR299:2, AR256:2, AR096:2, AR246:2, AR316:2, AR216:2, AR221:2, AR232:2, AR210:2, AR185:1, AR055:1, AR260:1, AR311:1, AR089:1, AR060:1,

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27	HCFMV71	526599	37	AR241:76, AR313:37, AR184:27, AR052:20, AR039:18, AR192:16, AR269:15, AR186:15, AR300:15, AR259:14, AR270:14, AR198:14, AR265:14, AR182:14, AR292:14, AR096:14, AR312:14, AR185:13, AR229:13, AR299:13, AR275:13, AR247:12, AR204:12, AR089:12, AR162:12, AR161:12, AR240:12, AR177:12, AR163:12, AR285:11, AR243:11, AR218:11, AR053:11, AR273:11, AR249:11, AR233:10, AR298:10, AR248:10, AR194:10, AR238:10, AR267:10, AR258:10, AR271:10, AR293:10, AR286:10, AR310:10, AR268:10, AR274:9, AR296:9, AR175:9, AR244:8, AR226:8, AR219:8, AR104:8, AR033:8, AR284:8, AR290:8, AR315:8, AR213:8, AR251:8, AR282:7, AR277:7, AR234:7, AR314:7, AR237:7, AR202:7, AR289:6, AR256:6, AR294:6, AR316:6, AR179:6, AR291:6, AR206:6, AR231:6, AR178:6, AR264:6, AR309:6, AR227:6, AR180:6, AR263:6, AR253:6, AR183:5, AR060:5, AR280:5, AR266:5, AR173:4, AR176:4, AR181:4, AR174:4, AR295:4, AR061:4, AR055:4, AR242:4, AR205:3, AR236:3, AR283:3, AR165:3, AR196:3, AR232:3, AR164:3, AR230:3, AR246:3, AR272:3, AR166:3, AR239:3, AR281:3, AR171:3, AR228:2, AR288:2, AR225:2, AR193:2, AR257:2, AR287:2, AR311:2, AR223:2, AR262:2, AR212:2, AR188:2, AR245:2, AR261:2, AR189:2, AR191:1, AR200:1, AR297:1, AR210:1, AR199:1 H0542:3, S6028:2, L0745:2, S0360:1, S0046:1, S0222:1, H0497:1, H0486:1, H0013:1, S0010:1, H0052:1, S0406:1 and L0439:1.
28	HCNSD93	630649	38	AR309:31, AR311:23, AR308:18, AR312:17, AR313:9, AR264:6, AR053:6, AR263:6, AR170:6, AR198:5, AR096:5, AR207:5, AR161:5, AR162:5, AR192:5, AR197:5, AR214:5, AR089:5, AR235:4, AR163:4, AR165:4, AR240:4, AR166:4, AR246:4, AR164:4, AR261:4, AR253:4, AR277:4, AR176:4, AR212:4, AR272:4, AR195:4, AR274:4, AR252:4, AR245:4, AR271:4, AR270:4, AR223:4, AR213:4, AR316:4, AR217:4, AR039:4, AR282:4, AR177:4, AR230:3, AR193:3, AR222:3, AR178:3, AR104:3, AR224:3, AR289:3, AR183:3, AR295:3, AR290:3, AR286:3, AR237:3, AR297:3, AR275:3, AR288:3, AR268:3, AR200:3, AR238:3, AR060:3, AR234:3, AR180:3, AR226:3, AR247:3, AR239:2, AR291:2, AR201:2, AR216:2, AR269:2, AR285:2, AR033:2, AR228:2, AR174:2, AR262:2, AR229:2, AR181:2, AR055:2, AR243:2, AR185:2, AR267:2, AR232:2, AR300:2, AR205:2, AR221:2, AR182:2, AR231:2, AR293:2, AR233:2, AR299:2, AR287:2, AR227:2, AR188:2, AR175:2, AR061:2, AR283:2, AR294:2, AR296:2, AR203:2, AR257:2, AR196:2, AR266:1, AR258:1, AR171:1, AR225:1, AR190:1, AR219:1, AR254:1, AR179:1, AR255:1, AR210:1, AR168:1 S0358:11, S0408:4, S0376:2, S0444:2, H0597:2, H0231:2, L0764:2, L0771:2, S0354:1, H0085:1, H0154:1, S0374:1, S0404:1 and H0423:1.
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30	HCWKCI5	553621	40		AR313:9, AR164:8, AR165:8, AR166:8, AR163:7, AR161:7, AR162:7, AR089:6, AR039:5, AR173:5, AR096:5, AR180:5, AR192:4, AR263:4, AR299:4, AR282:4, AR242:4, AR053:4, AR178:4, AR175:4, AR247:4, AR269:4, AR296:4, AR257:3, AR212:3, AR174:3, AR240:3, AR262:3, AR196:3, AR274:3, AR312:3, AR234:3, AR229:3, AR199:3, AR243:3, AR264:3, AR185:3, AR300:3, AR179:3, AR311:3, AR191:3, AR293:3, AR181:3, AR272:3, AR297:3, AR213:3, AR171:3, AR270:3, AR183:3, AR238:3, AR236:3, AR316:3, AR060:3, AR308:3, AR294:3, AR266:3, AR226:3, AR177:3, AR258:3, AR285:2, AR104:2, AR233:2, AR172:2, AR193:2, AR197:2, AR291:2, AR231:2, AR188:2, AR219:2, AR255:2, AR275:2, AR189:2, AR237:2, AR290:2, AR295:2, AR287:2, AR277:2, AR218:2, AR267:2, AR182:2, AR228:2, AR268:2, AR204:2, AR190:2, AR246:2, AR239:2, AR232:2, AR261:2, AR223:2, AR201:2, AR217:2, AR195:2, AR260:1, AR200:1, AR170:1, AR286:1, AR216:1, AR288:1, AR222:1, AR227:1, AR230:1 H0305:2 and H0589:1.
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35	HDPBA28	1062783	45	AR249:72, AR213:48, AR253:40, AR096:37, AR052:37, AR263:33, AR053:32, AR212:31, AR265:27, AR184:26,

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36	HDPCO25	460682	46	AR060:2, AR055:2, AR282:2 H0521:2, H0445:2, H0394:1, H0747:1, H0581:1, L0761:1 and L0750:1.
37	HDPCY37	837699	47	AR215:26, AR214:25, AR263:23, AR197:22, AR207:22, AR217:19, AR195:19, AR212:19, AR169:19, AR222:18, AR168:18, AR269:17, AR243:17, AR216:16, AR172:16, AR264:16, AR225:16, AR171:16, AR224:16, AR223:16, AR311:16, AR221:15, AR253:15, AR165:15, AR198:15, AR246:15, AR164:15, AR192:15, AR277:14, AR240:14, AR170:14, AR166:14, AR162:14, AR161:14, AR213:14, AR163:13, AR096:13, AR245:13, AR089:13, AR299:13, AR242:13, AR309:12, AR183:12, AR316:12, AR308:12, AR219:12, AR193:12, AR312:12, AR201:12, AR235:12, AR313:12, AR250:12, AR196:11, AR282:11, AR205:11, AR283:11, AR053:11, AR236:11, AR275:11, AR291:10, AR295:10, AR272:10, AR252:10, AR218:10, AR270:10, AR185:10, AR288:10, AR268:10, AR261:10, AR039:10, AR297:9, AR199:9, AR247:9, AR285:9, AR173:9, AR033:9, AR060:9, AR191:9, AR177:9, AR174:9, AR175:9, AR290:9, AR181:8, AR300:8, AR238:8, AR211:8, AR210:8, AR286:8, AR176:8, AR188:8, AR182:8, AR287:8, AR296:8, AR271:8, AR293:8, AR231:8, AR061:7, AR055:7, AR200:7, AR180:7, AR254:7, AR104:7, AR289:7, AR204:7, AR267:6, AR239:6, AR190:6, AR234:6, AR229:6, AR255:6, AR232:6, AR203:5, AR294:5, AR233:5, AR228:4, AR179:6, AR258:6, AR256:6, AR257:6, AR229:6, AR255:6, AR232:6, AR203:5, AR294:5, AR233:5, AR228:4, AR227:4, AR237:4, AR260:4, AR230:4 S0440:6, L0766:4, L3659:3, H0052:3, L0662:3, L0776:3, L0666:3, L0665:3, H0521:3, S0476:2, H0438:2, H0581:2, H0263:2, H0494:2, L0763:2, L0770:2, L0769:2, L0649:2, L0659:2, L0664:2, L2261:2, L3829:2, L0748:2, L0439:2, L0747:2, S0436:2, H0265:1, H0556:1, S0040:1, H0717:1, S0444:1, S0278:1, H0415:1, H0403:1, H0643:1, S0280:1, H0575:1, H0194:1, H0309:1, H0545:1, H0046:1, L0157:1, H0375:1, L0483:1, H0553:1, H0412:1, H0646:1, S0002:1, L0796:1, L3905:1, L0644:1, L0764:1, L0774:1, L0376:1, L0806:1, L0654:1, L0807:1, L0383:1, L3841:1, L2651:1, L2263:1, L2260:1, L2262:1, S0126:1, H0684:1, L0435:1, H0478:1, S0028:1, L0751:1, L0754:1, L0749:1, L0750:1, L0779:1, H0543:1, H0423:1 and L3837:1.
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39	HDPND46	637586	49	<p>AR272:69, AR212:53, AR214:43, AR311:39, AR274:36, AR245:35, AR165:33, AR216:32, AR308:32, AR166:31, AR161:30, AR162:30, AR217:29, AR264:29, AR163:29, AR222:28, AR164:28, AR215:27, AR309:27, AR171:26, AR223:25, AR053:25, AR252:23, AR224:23, AR168:23, AR174:22, AR225:21, AR169:21, AR205:21, AR213:21, AR195:21, AR312:20, AR197:20, AR172:19, AR263:18, AR275:18, AR247:17, AR254:17, AR221:17, AR170:17, AR313:15, AR185:15, AR189:15, AR199:15, AR236:15, AR188:14, AR242:14, AR201:14, AR250:13, AR246:13, AR193:13, AR288:12, AR190:12, AR297:11, AR230:11, AR179:11, AR253:11, AR096:10, AR243:10, AR240:10, AR239:10, AR262:9, AR177:9, AR089:9, AR300:9, AR255:9, AR194:9, AR287:9, AR290:9, AR060:9, AR173:9, AR291:9, AR238:9, AR203:8, AR257:8, AR271:8, AR178:8, AR296:8, AR200:8, AR232:8, AR204:8, AR289:8, AR299:8, AR295:8, AR293:8, AR231:8, AR261:8, AR282:8, AR316:8, AR234:8, AR265:8, AR285:7, AR191:7, AR226:7, AR277:7, AR181:7, AR233:7, AR061:7, AR180:6, AR198:6, AR192:6, AR237:6, AR210:6, AR283:6, AR270:6, AR039:6, AR228:6, AR207:6, AR294:6, AR280:6, AR269:6, AR229:5, AR186:5, AR315:5, AR266:5, AR183:5, AR033:5, AR267:5, AR268:5, AR104:5, AR211:5, AR286:5, AR176:5, AR227:5, AR298:4, AR175:4,</p>
40	HDPOH06	683371	50	<p>AR272:69, AR212:53, AR214:43, AR311:39, AR274:36, AR245:35, AR165:33, AR216:32, AR308:32, AR166:31, AR161:30, AR162:30, AR217:29, AR264:29, AR163:29, AR222:28, AR164:28, AR215:27, AR309:27, AR171:26, AR223:25, AR053:25, AR252:23, AR224:23, AR168:23, AR174:22, AR225:21, AR169:21, AR205:21, AR213:21, AR195:21, AR312:20, AR197:20, AR172:19, AR263:18, AR275:18, AR247:17, AR254:17, AR221:17, AR170:17, AR313:15, AR185:15, AR189:15, AR199:15, AR236:15, AR188:14, AR242:14, AR201:14, AR250:13, AR246:13, AR193:13, AR288:12, AR190:12, AR297:11, AR230:11, AR179:11, AR253:11, AR096:10, AR243:10, AR240:10, AR239:10, AR262:9, AR177:9, AR089:9, AR300:9, AR255:9, AR194:9, AR287:9, AR290:9, AR060:9, AR173:9, AR291:9, AR238:9, AR203:8, AR257:8, AR271:8, AR178:8, AR296:8, AR200:8, AR232:8, AR204:8, AR289:8, AR299:8, AR295:8, AR293:8, AR231:8, AR261:8, AR282:8, AR316:8, AR234:8, AR265:8, AR285:7, AR191:7, AR226:7, AR277:7, AR181:7, AR233:7, AR061:7, AR180:6, AR198:6, AR192:6, AR237:6, AR210:6, AR283:6, AR270:6, AR039:6, AR228:6, AR207:6, AR294:6, AR280:6, AR269:6, AR229:5, AR186:5, AR315:5, AR266:5, AR183:5, AR033:5, AR267:5, AR268:5, AR104:5, AR211:5, AR286:5, AR176:5, AR227:5, AR298:4, AR175:4,</p>

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42	HDPSP54 HDPVH60	502472 796865	213 52	<p>AR263:12, AR265:9, AR311:8, AR312:8, AR264:7, AR308:7, AR161:7, AR162:7, AR052:7, AR163:7, AR195:7, AR212:7, AR165:6, AR197:6, AR164:6, AR053:6, AR242:6, AR193:6, AR166:6, AR203:6, AR245:5, AR180:5, AR191:5, AR310:5, AR096:5, AR196:5, AR287:5, AR199:5, AR188:5, AR253:4, AR213:4, AR309:4, AR174:4, AR262:4, AR200:4, AR257:4, AR201:4, AR190:4, AR183:4, AR288:4, AR178:4, AR239:4, AR272:4, AR236:4, AR261:4, AR204:4, AR282:4, AR255:4, AR228:4, AR275:4, AR176:4, AR244:4, AR060:4, AR297:3, AR189:3, AR249:3, AR172:3, AR233:3, AR179:3, AR175:3, AR207:3, AR177:3, AR295:3, AR039:3, AR198:3, AR229:3, AR294:3, AR173:3, AR286:3, AR254:3, AR248:3, AR230:3, AR184:3, AR227:3, AR270:3, AR293:3, AR240:3, AR182:3, AR313:3, AR246:3, AR033:2, AR234:2, AR316:2, AR274:2, AR089:2, AR267:2, AR226:2, AR185:2, AR300:2, AR269:2, AR192:2, AR285:2, AR232:2, AR299:2, AR205:2, AR268:2, AR247:2, AR104:2, AR237:2, AR271:2, AR221:2, AR290:2, AR061:2, AR231:2, AR181:2, AR252:2, AR260:2, AR277:2, AR281:2, AR289:2, AR243:1, AR266:1, AR055:1, AR291:1, AR296:1, AR168:1, AR211:1, AR235:1, AR280:1, AR292:1, AR251:1, AR219:1, AR194:1, H0457:6, H0436:4, L0761:3, L0655:3, L0749:3, S0276:3, H0716:2, H0657:2, H0492:2, H0069:2, H0050:2, H0271:2, L0764:2, L0771:2, L0766:2, L0774:2, L0775:2, H0521:2, L0751:2, L0777:2, H0423:2, S0114:1, S0134:1, H0650:1, L0808:1, H0254:1, S0376:1, S0360:1, H0580:1, H0600:1, H0586:1, H0587:1,</p>

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	HDPWN93	905983	215	
44	HIDQHD03	1309175	54	AR206:6, AR263:4, AR244:3, AR273:3, AR310:2, AR215:2, AR250:2, AR169:2, AR243:2, AR171:2, AR282:2, AR216:2, AR253:2, AR285:2, AR247:2, AR183:2, AR277:2, AR060:2, AR212:1, AR217:1, AR238:1, AR312:1, AR186:1, AR271:1, AR266:1, AR055:1, AR255:1, AR262:1, AR311:1, AR289:1, AR231:1, AR296:1, AR257:1, AR290:1, AR204:1, AR096:1, AR089:1, AR227:1 L0766:5, L0779:2, T0082:1 and L0807:1.
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45	HE2EN04	545008	55	AR309:12, AR264:11, AR308:9, AR263:9, AR311:8, AR312:6, AR210:6, AR225:6, AR207:5, AR053:5, AR245:5, AR200:5, AR313:4, AR272:4, AR282:4, AR217:4, AR271:4, AR223:4, AR201:4, AR183:4, AR212:4, AR196:4, AR193:4, AR246:4, AR270:3, AR274:3, AR203:3, AR162:3, AR161:3, AR163:3, AR267:3, AR176:3, AR205:3, AR195:3, AR172:3, AR261:3, AR096:3, AR165:3, AR197:3, AR164:3, AR268:2, AR218:2, AR255:2, AR177:2, AR204:2, AR188:2, AR168:2, AR199:2, AR175:2, AR166:2, AR316:2, AR060:2, AR216:2, AR236:2, AR089:2, AR266:2, AR288:2, AR171:2, AR213:2, AR178:2, AR228:2, AR262:2, AR290:2, AR231:2, AR179:2, AR233:2, AR185:2, AR239:2, AR296:2, AR229:2, AR234:2, AR182:2, AR289:2, AR285:2, AR277:1, AR224:1, AR181:1, AR293:1, AR191:1, AR237:1, AR227:1, AR219:1, AR286:1, AR173:1, AR291:1, AR269:1, AR295:1, AR190:1, AR258:1, AR055:1, AR294:1, AR211:1, AR061:1, AR238:1, AR252:1, AR247:1, AR297:1, AR283:1, AR299:1,

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47	HE8QV67 HE8UB86	1050077 834913	217 57	AR266:6, AR176:5, AR183:5, AR192:5, AR182:4, AR215:4, AR181:4, AR274:4, AR055:4, AR235:4, AR269:4, AR223:4, AR217:4, AR236:4, AR228:4, AR060:4, AR178:3, AR224:3, AR257:3, AR165:3, AR229:3, AR270:3, AR233:3, AR168:3, AR161:3, AR268:3, AR166:3, AR162:3, AR163:3, AR237:3, AR164:3, AR214:3, AR253:3, AR261:3, AR267:3, AR225:3, AR179:3, AR177:3, AR293:3, AR180:3, AR296:3, AR212:3, AR247:3, AR175:3, AR191:2, AR289:2, AR238:2, AR282:2, AR222:2, AR231:2, AR291:2, AR294:2, AR196:2, AR039:2, AR277:2, AR262:2, AR199:2, AR245:2, AR240:2, AR255:2, AR295:2, AR288:2, AR216:2, AR297:2, AR089:2, AR061:2, AR200:2, AR239:2, AR174:2, AR201:2, AR309:2, AR188:2, AR190:2, AR300:2, AR203:2, AR271:2, AR230:2, AR285:2, AR234:2, AR316:2, AR189:2, AR299:2, AR226:2, AR185:2, AR227:2, AR286:2, AR275:2, AR193:2, AR172:2, AR096:2, AR232:2, AR104:2, AR313:2, AR221:2, AR195:2, AR290:2, AR283:2, AR258:1, AR287:1, AR219:1, AR264:1, AR173:1, AR312:1, AR218:1, AR210:1, AR169:1 H0030:2, H0624:1, H0013:1, L0769:1, L0803:1 and L0438:1.
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68	HHEMA75	494099	78	AR245:10, AR207:7, AR197:7, AR242:6, AR169:6, AR282:6, AR221:6, AR243:6, AR195:5, AR224:5, AR309:5, AR198:5, AR089:5, AR171:5, AR201:5, AR250:5, AR165:5, AR311:5, AR164:5, AR039:5, AR214:5, AR246:5, AR216:5, AR180:4, AR263:4, AR313:4, AR168:4, AR166:4, AR225:4, AR053:4, AR222:4, AR205:4, AR170:4, AR253:4, AR283:4, AR161:4, AR252:4, AR299:4, AR254:4, AR193:4, AR162:4, AR172:4, AR271:3, AR235:3, AR192:3, AR196:3, AR274:3, AR163:3, AR223:3, AR295:3, AR312:3, AR060:3, AR264:3, AR177:3, AR288:3, AR272:3, AR261:3, AR316:3, AR178:3, AR308:3, AR257:3, AR217:3, AR183:3, AR175:3, AR291:3, AR188:3, AR285:3, AR191:3, AR174:3, AR236:3, AR212:3, AR286:2, AR182:2, AR238:2, AR190:2, AR213:2, AR237:2, AR189:2, AR227:2, AR293:2, AR185:2, AR173:2, AR275:2, AR294:2, AR230:2, AR229:2, AR200:2, AR226:2,

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81	HKACB56	554616	91	AR271:11, AR242:9, AR216:8, AR253:7, AR214:7, AR205:7, AR195:6, AR165:6, AR207:6, AR296:6, AR164:6, AR198:6, AR089:6, AR254:6, AR224:6, AR250:6, AR166:6, AR217:6, AR309:6, AR212:6, AR245:6, AR192:6, AR263:6, AR215:6, AR221:6, AR312:5, AR162:5, AR196:5, AR308:5, AR161:5, AR096:5, AR299:5, AR163:5, AR246:5, AR213:5, AR313:5, AR243:5, AR053:5, AR193:5, AR222:5, AR264:5, AR223:5, AR311:5, AR060:5, AR204:4, AR197:4, AR188:4, AR274:4, AR261:4, AR175:4, AR201:4, AR172:4, AR285:4, AR189:4, AR316:4, AR039:4, AR169:4, AR171:4, AR173:4, AR300:4, AR268:4, AR282:4, AR176:4, AR199:4, AR104:4, AR033:4, AR168:4, AR235:4, AR190:4, AR200:4, AR240:4, AR295:3, AR288:3, AR257:3, AR277:3, AR252:3, AR291:3, AR297:3, AR203:3, AR238:3, AR286:3, AR177:3, AR294:3, AR174:3, AR289:3, AR191:3, AR183:3, AR210:3, AR283:3, AR185:3, AR180:3, AR255:3, AR178:3, AR247:3, AR290:3, AR262:3, AR269:3, AR230:3, AR293:3, AR270:3, AR287:2, AR226:2, AR181:2, AR258:2, AR275:2, AR219:2, AR267:2, AR218:2, AR239:2, AR179:2, AR232:2, AR234:2, AR272:2, AR237:2, AR229:2, AR231:2, AR061:2, AR233:2, AR236:2, AR228:2, AR182:2, AR227:1, AR256:1, AR266:1, AR170:1, L0794:9, L0777:6, L0809:4, L0779:4, L0731:4, L0766:3, L0666:3, L0663:3, L3825:3, H0547:3, S0444:2, L3459:2, L3480:2, L3817:2, L0483:2, L0770:2, L0521:2, L0768:2, L0803:2, L0775:2, L0805:2, L0661:2, L0665:2, H0144:2, L3827:2, L3828:2, H0658:2, H0670:2, S0406:2, L0439:2, L0754:2, L0749:2, L0756:2, H0543:2, H0556:1, H0657:1, H0662:1, S0360:1, L3262:1, L2799:1, H0411:1, S0278:1, H0443:1, H0550:1, L3816:1, T0039:1, L3499:1, L2647:1, H0013:1, H0427:1, H0575:1, S0474:1, H0052:1, H0591:1, H0038:1, H0040:1, H0616:1, H0264:1, H0494:1, S0440:1, H0649:1, L0598:1, H0529:1, L0369:1, L0640:1, L3904:1, L0662:1, L0804:1, L0375:1, L0378:1, L0806:1, L0653:1, L0776:1, L0807:1, L0788:1, L0664:1, L2259:1, L2654:1, L3812:1, S0126:1, H0689:1, H0435:1, H0539:1, H0696:1, S0176:1, H0555:1, H0785:1, L0747:1, L0755:1, L0757:1, L0758:1, L0608:1, L0362:1, S0026:1, S0424:1 and L3808:1.
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92	HLQDH79	588446	102	AR235:19, AR296:17, AR288:16, AR287:15, AR295:15, AR255:15, AR261:14, AR285:14, AR256:14, AR291:14, AR297:13, AR286:13, AR236:12, AR289:12, AR258:12, AR262:11, AR193:11, AR293:11, AR162:10, AR161:10, AR266:10, AR163:10, AR294:10, AR169:10, AR171:10, AR257:9, AR260:9, AR313:9, AR283:9, AR189:9, AR089:9, AR170:9, AR196:9, AR178:9, AR269:9, AR182:9, AR168:8, AR247:8, AR268:8, AR191:8, AR225:8, AR190:8, AR165:8, AR223:8, AR218:8, AR309:8, AR240:7, AR164:7, AR176:7, AR188:7, AR183:7, AR270:7, AR316:7, AR166:7, AR215:7, AR254:7, AR217:7, AR173:7, AR312:7, AR175:7, AR229:7, AR250:7, AR245:7, AR096:7, AR300:7, AR199:6, AR214:6, AR201:6, AR177:6, AR267:6, AR174:6, AR231:6, AR180:6, AR246:6, AR238:6, AR282:6, AR207:6, AR198:6, AR233:6, AR197:6, AR290:6, AR181:6, AR203:6, AR104:6, AR053:6, AR179:6,

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96	HLWCF05	460619	106	

97	HL YAF80	460622	107	<p>H0657:2, H0341:2, S0418:2, S0444:2, S0410:2, H0747:2, S0476:2, L3655:2, H0013:2, H0553:2, H0032:2, H0169:2, L0455:2, H0040:2, S0422:2, H0529:2, L0667:2, L0662:2, L0768:2, L0519:2, L0754:2, L0745:2, L0747:2, L0750:2, L0779:2, L0731:2, S0434:2, S0436:2, L0592:2, S0412:2, H0556:1, T0002:1, S0114:1, S0116:1, L0879:1, H0638:1, S0420:1, S0356:1, S0358:1, S0376:1, S0408:1, L1499:1, H0749:1, H0619:1, L2817:1, L3485:1, H0586:1, H0587:1, H0333:1, H0574:1, H0632:1, T0039:1, L1788:1, L1877:1, L0021:1, L0022:1, H0575:1, S0474:1, H0581:1, H0457:1, H0320:1, H0014:1, L0163:1, H0375:1, H0188:1, S0250:1, L0483:1, H0598:1, H0163:1, H0591:1, H0616:1, H0623:1, H0100:1, H0494:1, S0440:1, L0598:1, L0763:1, L0769:1, L0638:1, L0800:1, L0641:1, L0794:1, L0803:1, L0775:1, L0806:1, L0776:1, L0527:1, L0659:1, L0635:1, L0787:1, L0789:1, L0666:1, L0663:1, L0664:1, L0665:1, S0428:1, L2653:1, L2261:1, H0519:1, H0435:1, H0670:1, H0672:1, H0539:1, H0696:1, S0406:1, H0436:1, H0727:1, L0755:1, L0485:1, H0423:1 and H0506:1.</p>
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99	HM ADS41	596831	109	<p>AR218:19, AR219:19, AR283:12, AR096:12, AR313:11, AR316:10, AR240:10, AR300:9, AR185:9, AR055:9, AR277:9, AR039:8, AR089:8, AR282:8, AR060:8, AR299:7, AR104:7 L0794:4, L0375:3, H0575:2, L0800:2, L0789:2, H0556:1, H0662:1, S0418:1, H0619:1, H0549:1, H0590:1, H0052:1, H0083:1, H0266:1, H0286:1, H0644:1, S0036:1, H0433:1, H0412:1, H0413:1, T0042:1, S0144:1, S0142:1, S0344:1, L0770:1, L0761:1, L0774:1, H0518:1, L0777:1, L0758:1 and H0665:1.</p>

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	HMADU73	467053	238	
101	HMIAL37	603201	111	AR266:6, AR207:6, AR176:6, AR217:5, AR162:5, AR161:5, AR225:5, AR163:5, AR183:5, AR182:5, AR269:5, AR245:5, AR223:5, AR214:4, AR288:4, AR205:4, AR309:4, AR181:4, AR270:4, AR267:4, AR291:4, AR216:4, AR215:4, AR261:4, AR242:4, AR274:4, AR171:4, AR289:3, AR233:3, AR235:3, AR177:3, AR195:3, AR175:3, AR286:3, AR053:3, AR287:3, AR198:3, AR268:3, AR294:3, AR236:3, AR237:3, AR255:3, AR228:3, AR180:3, AR238:3, AR257:3, AR173:3, AR172:3, AR311:3, AR271:3, AR290:3, AR293:3, AR191:3, AR179:3, AR201:3, AR192:3, AR221:3, AR229:3, AR285:3, AR247:3, AR296:3, AR275:3, AR061:3, AR199:3, AR193:2, AR165:2, AR230:2, AR166:2, AR170:2, AR164:2, AR190:2, AR243:2, AR222:2, AR178:2, AR262:2, AR060:2, AR039:2, AR231:2, AR256:2, AR204:2, AR260:2, AR200:2, AR168:2, AR297:2, AR189:2, AR188:2, AR234:2, AR239:2, AR282:2, AR316:2, AR240:2, AR272:2, AR096:2, AR295:2, AR258:2, AR224:2, AR300:2, AR226:2, AR203:2, AR232:2, AR196:2, AR246:2, AR104:2, AR213:1, AR185:1, AR299:1, AR227:1, AR089:1, AR277:1, AR312:1, AR308:1, AR169:1, AR033:1, AR055:1, AR174:1 S0354:2, H0549:2, S0442:1, S0360:1, S0010:1, S0050:1, H0015:1, S6028:1, H0622:1, S0038:1, S0440:1, S0436:1 and L0596:1.
102	HMKCG09	548078	112	AR202:39, AR292:25, AR280:25, AR315:25, AR104:24, AR310:24, AR284:23, AR312:20, AR052:20, AR281:19, AR314:19, AR309:19, AR275:19, AR266:18, AR186:18, AR033:17, AR060:17, AR295:17, AR285:17, AR283:16, AR298:16, AR259:16, AR055:15, AR273:14, AR271:14, AR192:13, AR277:13, AR286:13, AR204:13, AR185:12, AR253:12, AR184:12, AR250:12, AR289:11, AR251:11, AR291:11, AR241:10, AR218:10, AR096:10, AR294:10, AR299:10, AR274:10, AR265:9, AR316:9, AR293:9, AR183:9, AR219:9, AR089:9, AR213:8, AR282:8, AR254:8, AR272:8, AR270:8, AR244:7, AR039:7, AR238:7, AR269:7, AR256:7, AR258:7, AR201:7, AR296:7, AR182:7, AR177:6, AR175:6, AR205:6, AR195:6, AR247:6, AR268:6, AR248:6, AR198:6, AR300:5, AR232:5, AR231:5, AR290:5, AR053:5, AR206:5, AR249:5, AR263:5, AR061:5, AR267:5, AR165:4, AR164:4, AR226:4, AR252:4, AR237:4, AR214:4, AR166:4, AR215:4, AR229:3, AR207:3, AR212:3, AR240:3, AR257:3, AR191:3, AR171:3, AR264:3, AR227:3, AR170:3, AR262:3, AR173:3, AR233:3, AR199:2, AR243:2, AR246:2, AR297:2, AR236:2, AR180:2, AR196:2, AR197:2, AR179:2, AR188:2, AR234:2, AR189:2, AR228:2, AR223:2, AR168:2, AR190:2, AR200:2, AR288:2, AR225:2, AR239:2, AR261:1, AR287:1, AR308:1, AR193:1, AR181:1, AR216:1, AR224:1, AR255:1, AR174:1 L0766:7, L0803:7, S0466:2, L0805:2, L3387:1, H0392:1, H0156:1, L0021:1, H0052:1, L0770:1, L0804:1, L0788:1, H0756:1, L0743:1, L0755:1, L0731:1 and L0361:1.
103	HMMAH60	562776	113	AR242:10, AR313:9, AR192:9, AR196:7, AR173:7, AR165:7, AR089:7, AR164:6, AR197:6, AR039:6, AR161:6,

104	HMQDT36	1309723	114	AR162:6, AR245:6, AR163:6, AR193:5, AR053:5, AR299:5, AR183:5, AR175:5, AR271:5, AR257:5, AR204:4, AR174:4, AR033:4, AR261:4, AR096:4, AR300:4, AR178:4, AR229:4, AR240:4, AR262:4, AR199:4, AR252:4, AR191:4, AR243:4, AR189:4, AR293:4, AR177:4, AR247:4, AR264:4, AR179:4, AR180:4, AR238:4, AR269:4, AR235:4, AR166:4, AR182:4, AR250:4, AR201:4, AR195:4, AR203:4, AR170:4, AR218:4, AR213:4, AR296:3, AR060:3, AR316:3, AR258:3, AR275:3, AR200:3, AR236:3, AR185:3, AR270:3, AR176:3, AR285:3, AR234:3, AR297:3, AR272:3, AR312:3, AR188:3, AR277:3, AR104:3, AR221:3, AR198:3, AR230:3, AR226:3, AR266:3, AR210:3, AR233:3, AR237:3, AR268:3, AR239:3, AR216:3, AR231:3, AR181:3, AR288:3, AR222:3, AR290:3, AR211:3, AR294:2, AR190:2, AR267:2, AR286:2, AR282:2, AR274:2, AR291:2, AR171:2, AR295:2, AR228:2, AR287:2, AR207:2, AR246:2, AR308:2, AR219:2, AR255:2, AR212:2, AR223:2, AR227:2, AR260:2, AR225:2, AR232:2, AR215:2, AR217:1, AR253:1, AR055:1, AR061:1 L0547:1 and H0444:1.
104	HMQDT36	1309723	114	AR218:25, AR219:21, AR096:17, AR039:13, AR316:13, AR089:12, AR299:9, AR055:9, AR282:9, AR060:8, AR252:7, AR185:7, AR313:7, AR277:6, AR240:6, AR300:6, AR104:5, AR283:4, AR263:4, AR164:4, AR192:4, AR243:4, AR253:3, AR170:3, AR212:3, AR269:3, AR224:3, AR286:3, AR204:3, AR264:3, AR180:3, AR296:2, AR293:2, AR247:2, AR246:2, AR196:2, AR168:2, AR295:2, AR165:2, AR289:2, AR176:2, AR214:2, AR053:2, AR271:2, AR175:2, AR268:2, AR171:2, AR234:2, AR222:2, AR178:2, AR275:2, AR213:1, AR182:1, AR173:1, AR267:1, AR285:1, AR225:1, AR183:1, AR189:1, AR195:1, AR201:1, AR308:1, AR312:1, AR239:1, AR174:1, AR181:1, AR193:1, AR172:1 L0754:10, L0748:9, L0770:8, H0521:8, S0003:7, S0356:5, L0751:5, S0436:5, S0358:4, S0360:4, H0494:4, L0764:4, L0803:4, L0731:4, H0580:3, H0615:3, H0591:3, H0040:3, H0623:3, S0422:3, L0771:3, L0776:3, L0666:3, S0406:3, L0752:3, S0434:3, H0542:3, S0212:2, H0255:2, H0638:2, S0418:2, L0005:2, S0442:2, S0376:2, S0408:2, S0045:2, S0476:2, H0497:2, H0231:2, H0266:2, H0179:2, S0214:2, H0622:2, H0124:2, H0551:2, S0440:2, L0805:2, L0809:2, S0374:2, H0547:2, H0660:2, H0648:2, H0709:2, L0740:2, L0759:2, H0445:2, L0596:2, L0599:2, H0506:2, H0556:1, T0002:1, H0713:1, H0717:1, L3404:1, T0049:1, S0218:1, H0583:1, H0341:1, L3658:1, S0354:1, S0444:1, H0730:1, H0741:1, H0208:1, H0749:1, H0351:1, H0549:1, H0550:1, S0222:1, H0331:1, H0574:1, H0250:1, H0069:1, H0635:1, H0427:1, S0280:1, T0071:1, H0263:1, H0596:1, H0046:1, H0123:1, H0014:1, H0354:1, H0375:1, H0428:1, H0039:1, T0023:1, L0483:1, T0006:1, H0031:1, L0142:1, L0143:1, L0055:1, H0673:1, S0366:1, H0598:1, H0038:1, H0634:1, H0269:1, H0059:1, H0429:1, H0561:1, S0438:1, H0509:1, S0150:1, H0646:1, H0652:1, S0142:1, S0210:1, S0002:1, S0426:1, H0529:1, L0637:1, L0800:1, L0662:1, L0767:1, L0387:1, L0766:1, L0388:1, L0522:1, L0804:1, L0774:1, L0775:1, L0378:1, L0806:1, L0652:1, L0654:1, L0655:1, L0661:1, L0657:1, L0659:1, L0647:1, L5623:1, L0664:1, S0052:1, S0428:1, L3819:1, H0701:1, T0068:1, L3811:1, S0126:1, H0682:1, H0659:1, H0670:1, S0378:1, H0518:1, S0152:1, H0696:1, S0146:1, L0758:1, L0608:1, H0667:1, S0192:1, S0242:1, S0194:1, H0543:1, H0423:1 and H0422:1.
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105	HMSHS36	1127691	115	AR039:6, AR055:5, AR218:5, AR060:5, AR300:5, AR185:4, AR313:4, AR299:4, AR240:4, AR104:3, AR316:3, AR096:3, AR282:3, AR089:3, AR283:2, AR277:1 S0002:1
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106	HMSIU68	427121	116	AR089:10, AR055:9, AR060:7, AR039:7, AR185:7, AR313:7, AR300:6, AR218:6, AR316:5, AR240:5, AR104:5,

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108	HMTAD67	588447	118	AR313:17, AR196:14, AR173:13, AR165:11, AR161:10, AR164:10, AR162:10, AR166:10, AR242:10, AR163:10, AR180:10, AR096:10, AR089:9, AR240:9, AR262:8, AR178:8, AR247:8, AR258:8, AR200:8, AR296:8, AR185:8, AR191:8, AR175:8, AR300:7, AR234:7, AR179:7, AR238:7, AR236:7, AR257:7, AR183:7, AR264:7, AR199:7, AR213:7, AR169:7, AR229:7, AR181:6, AR168:6, AR299:6, AR219:6, AR297:6, AR287:6, AR293:6, AR170:6, AR060:6, AR218:6, AR203:6, AR177:6, AR182:6, AR192:6, AR316:6, AR215:6, AR171:6, AR104:5, AR261:5, AR174:5, AR255:5, AR291:5, AR274:5, AR263:5, AR294:5, AR212:5, AR188:5, AR312:5, AR275:5, AR039:5, AR245:5, AR207:5, AR226:5, AR230:5, AR282:5, AR233:5, AR053:5, AR269:5, AR270:5, AR189:5, AR033:5, AR223:5, AR285:4, AR237:4, AR231:4, AR260:4, AR277:4, AR268:4, AR204:4, AR214:4, AR254:4, AR286:4, AR228:4, AR290:4, AR176:4, AR235:4, AR272:4, AR311:4, AR239:3, AR288:3, AR295:3, AR193:3, AR211:3, AR309:3, AR217:3, AR308:3, AR266:3, AR283:3, AR252:3, AR172:3, AR267:3, AR198:3, AR227:3, AR246:3, AR190:3, AR222:2, AR289:2, AR210:2, AR271:2, AR061:2, AR205:2, AR225:2, AR201:1, AR256:1, AR232:1, AR195:1, AR216:1, AR243:1, L0439:9, S0440:8, L0438:7, H0521:5, S0040:3, H0556:2, S0222:2, H0457:2, H0024:2, H0687:2, H0031:2, H0264:2, H0488:2, H0494:2, H0529:2, L5622:2, L0663:2, L0751:2, L0596:2, H0265:1, L3644:1, S0134:1, H0484:1, H0661:1, H0638:1, S0418:1, S0442:1, L3713:1, H0733:1, S0045:1, H0749:1, H0261:1, H0370:1, H0590:1, H0618:1, H0253:1, H0194:1, H0083:1, H0510:1, H0594:1, H0266:1, H0644:1, H0032:1, H0087:1, H0272:1, H0268:1, H0623:1, H0102:1, H0561:1, H0509:1, L3904:1, L3905:1, L0651:1, L0526:1, L2262:1, L3825:1, L3827:1, H0593:1, L0602:1, H0518:1, H0436:1, H0478:1, S3012:1, S0028:1, L0731:1, S0436:1, L0605:1, L0601:1, H0667:1, S0194:1, H0543:1, H0506:1 and H0008:1.
109	HMWFO02	1352198	119	AR173:11, AR258:8, AR178:8, AR175:8, AR313:7, AR196:7, AR183:7, AR262:7, AR219:7, AR218:7, AR257:6, AR182:6, AR293:6, AR191:6, AR169:6, AR179:6, AR236:6, AR240:6, AR180:6, AR200:6, AR174:6, AR199:6, AR229:5, AR235:5, AR176:5, AR270:5, AR261:5, AR181:5, AR285:5, AR247:5, AR161:5, AR299:5, AR233:5, AR162:5, AR163:5, AR288:5, AR269:5, AR268:5, AR287:5, AR235:5, AR260:5, AR226:4, AR228:4, AR096:4, AR234:4, AR189:4, AR177:4, AR290:4, AR296:4, AR294:4, AR297:4, AR286:4, AR300:4, AR295:4, AR165:4,

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	HPWAY46	789574	267	
140	HPWAZ95	413270	150	AR296:12, AR295:12, AR161:11, AR162:11, AR163:11, AR089:9, AR291:9, AR165:9, AR164:9, AR166:8, AR261:8, AR104:8, AR313:8, AR270:8, AR269:7, AR235:7, AR309:7, AR192:7, AR178:7, AR180:7, AR285:7, AR242:7, AR176:7, AR271:7, AR253:7, AR033:7, AR297:6, AR182:6, AR053:6, AR228:6, AR268:6, AR183:6, AR266:6, AR294:6, AR255:6, AR293:5, AR175:5, AR264:5, AR181:5, AR236:5, AR267:5, AR191:5, AR060:5, AR290:5, AR243:5, AR173:5, AR287:5, AR229:5, AR282:5, AR257:5, AR096:5, AR193:5, AR316:5, AR289:5, AR188:5,

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182	HUKBT29	694590	192	AR180:4, AR172:3, AR225:3, AR271:2, AR242:2, AR170:2, AR221:2, AR275:2, AR183:2, AR283:2, AR181:2, AR264:2, AR214:2, AR213:2, AR257:1, AR277:1, AR195:1, AR171:1, AR205:1, AR222:1, AR261:1, AR164:1, AR176:1, S0366:3, H0599:2, H0059:2, H0547:2, L0604:2, H0543:2, H0149:1, L0460:1, S0430:1, H0255:1, H0728:1, H0002:1, H0051:1, S0364:1, H0116:1, L5575:1, L0794:1, L0803:1, S0428:1, S0330:1, H0522:1, H0555:1, L0747:1, L0777:1, L0485:1, L0366:1 and S0446:1.
183	HUSBA88	895435	193	AR251:27, AR250:24, AR273:24, AR264:23, AR243:22, AR263:21, AR253:20, AR245:18, AR265:17, AR309:17, AR254:16, AR312:15, AR310:14, AR240:13, AR308:12, AR213:11, AR212:11, AR275:11, AR180:10, AR096:10, AR242:10, AR053:10, AR162:10, AR052:10, AR161:10, AR268:10, AR163:9, AR197:9, AR313:9, AR272:9, AR165:8, AR248:8, AR164:8, AR271:8, AR274:8, AR247:8, AR290:8, AR249:8, AR311:8, AR166:8, AR246:8, AR195:7, AR299:7, AR089:7, AR200:7, AR270:7, AR193:7, AR241:7, AR183:7, AR176:7, AR199:7, AR282:7, AR201:7, AR196:7, AR190:7, AR178:6, AR198:6, AR300:6, AR175:6, AR189:6, AR191:6, AR316:6, AR266:6, AR192:6, AR207:6, AR267:6, AR039:6, AR179:5, AR181:5, AR173:5, AR188:5, AR218:5, AR215:5, AR269:5, AR203:5, AR174:5, AR186:5, AR060:5, AR219:5, AR225:5, AR280:4, AR177:4, AR185:4, AR170:4, AR283:4, AR292:4, AR182:4, AR314:4, AR284:4, AR104:4, AR205:4, AR235:4, AR221:3, AR229:3, AR289:3, AR206:3, AR291:3, AR204:3, AR234:3, AR259:3, AR256:3, AR261:3, AR262:3, AR231:3, AR293:3, AR257:3, AR228:3, AR296:3, AR295:3, AR033:3, AR298:3, AR287:3, AR238:3, AR285:3, AR184:3, AR224:2, AR315:2, AR237:2, AR168:2, AR061:2, AR286:2, AR288:2, AR211:2, AR217:2, AR252:2, AR236:2, AR239:2, AR171:2, AR294:2, AR210:2, AR172:2, AR055:2, AR216:2, AR227:2, AR297:2, AR232:2, AR258:2, AR244:2, AR230:2, AR226:2, AR233:1, AR281:1, AR222:1, AR169:1, AR214:1, AR260:1, L0747:9, H0251:8, L0742:7, L0748:7, L0439:7, S0360:6, L0754:6, L0759:6, H0013:5, H0553:5, H0059:5, L0770:5, L0771:5, L0809:5, L0664:5, H0520:5, L0752:5, S0140:4, H0052:4, H0124:4, H0616:4, H0529:4, L0768:4, L0794:4, L0775:4, L0378:4, L0665:4, H0144:4, H0658:4, L0602:4, S0408:3, S0132:3, H0617:3, H0100:3, L0639:3, L5566:3, L0659:3, L0666:3, H0670:3, S0206:3, L0751:3, L0731:3, L0758:3, L0605:3, S0114:2, S0442:2, S0444:2, L0717:2, H0550:2, S0222:2, H0370:2, H0392:2, H0455:2, H0333:2, H0486:2, H0253:2, H0545:2, H0150:2, H0213:2, H0644:2, H0135:2, S0038:2, L0351:2, H0494:2, S0426:2, L0763:2, L0769:2, L0761:2, L0764:2, L0773:2, L0803:2, L0527:2, L0657:2, L0783:2, L0663:2, H0547:2, S0126:2, H0684:2, H0672:2, H0651:2, S0406:2, H0479:2, S0028:2, L0740:2, L0749:2, L0750:2, L0777:2,

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187	HWHGP71	995431	197	AR244:4, AR169:4, AR170:4, AR215:3, AR252:3, AR250:3, AR180:3, AR310:3, AR184:2, AR207:2, AR251:2, AR195:2, AR264:2, AR311:2, AR214:2, AR282:1, AR313:1, AR165:1, AR312:1, AR171:1, AR270:1, AR269:1, AR263:1, AR212:1, AR166:1, AR240:1, AR223:1, AR202:1, AR247:1, AR239:1, AR238:1, AR309:1, AR096:1, AR204:1, AR168:1, AR257:1 H0586:1, H0457:1, H0634:1 and H0521:1.
	HWHGP71	839250	294	
188	HWHQ55	762842	198	AR274:28, AR247:26, AR272:21, AR096:21, AR283:21, AR213:20, AR312:19, AR240:19, AR161:18, AR162:18, AR163:18, AR172:17, AR311:17, AR216:17, AR309:17, AR313:17, AR291:17, AR254:17, AR308:17, AR165:16, AR245:16, AR164:15, AR089:15, AR166:15, AR263:14, AR282:13, AR266:13, AR205:13, AR173:13, AR264:13, AR171:13, AR169:13, AR168:13, AR170:13, AR222:13, AR212:13, AR270:12, AR183:12, AR214:12, AR039:11, AR223:11, AR179:11, AR217:11, AR246:11, AR316:11, AR224:11, AR290:10, AR175:10, AR210:10, AR178:10, AR243:10, AR188:10, AR269:10, AR271:10, AR288:10, AR242:9, AR277:9, AR189:9, AR289:9, AR176:9, AR268:9, AR181:9, AR180:9, AR255:9, AR250:9, AR221:9, AR299:9, AR253:9, AR275:9, AR293:9, AR297:9, AR262:8, AR267:8, AR190:8, AR296:8, AR177:8, AR235:8, AR225:8, AR174:8, AR053:8, AR300:8, AR257:8, AR211:8, AR055:8, AR197:8, AR219:8, AR185:8, AR218:8, AR199:8, AR060:7, AR236:7, AR195:7, AR287:7, AR261:7, AR193:7, AR215:7, AR061:7, AR295:7, AR258:7, AR285:7, AR196:7, AR192:6, AR191:6, AR256:6, AR204:6, AR104:6, AR207:6, AR260:6, AR294:6, AR201:6, AR182:6, AR286:6, AR231:5, AR198:5, AR200:5, AR033:5, AR203:5, AR252:5, AR233:4, AR237:4, AR234:4, AR229:4, AR227:4, AR238:4, AR232:4, AR239:3, AR230:3, AR226:3, AR228:3 L0439:6, H0620:4, L0758:2, S0040:1, S0282:1, H0661:1, H0619:1, H0549:1, H0587:1, H0013:1, L0021:1, H0230:1, H0009:1, H0373:1, H0135:1, L0770:1, L0769:1, L0776:1, L0659:1, L0783:1, H0144:1, H0519:1, H0593:1, H0682:1, H0659:1, L0751:1, L0753:1 and L0759:1.
189	HWLEV32	1032602	199	AR039:14, AR313:12, AR096:8, AR089:7, AR299:7, AR185:5, AR277:5, AR282:5, AR316:4, AR300:4, AR104:4, AR198:4, AR182:3, AR060:3, AR240:3, AR246:3, AR178:3, AR215:3, AR225:3, AR263:2, AR216:2, AR218:2, AR201:2, AR274:2, AR270:2, AR227:2, AR243:2, AR165:2, AR164:2, AR247:2, AR055:2, AR269:2, AR257:2, AR179:2, AR242:2, AR033:2, AR309:2, AR311:2, AR199:2, AR205:2, AR224:1, AR200:1, AR275:1, AR191:1, AR291:1, AR168:1, AR289:1, AR236:1, AR219:1, AR193:1, AR230:1, AR312:1, AR308:1, AR192:1 L0731:3, S0194:3, H0392:2, H0031:2, H0644:2, H0494:2, L0794:2, L0803:2, L0666:2, S0330:2, S3014:2, L0747:2, L0777:2,

					L0758:2, S0026:2, H0556:1, H0717:1, S0298:1, S0282:1, L3658:1, S0418:1, S0356:1, S0354:1, S0444:1, S0360:1, H0722:1, L0717:1, H0431:1, S0346:1, H0421:1, H0052:1, H0150:1, S0388:1, H0083:1, H0252:1, H0604:1, H0030:1, H0412:1, L0769:1, L0662:1, L0768:1, L0375:1, L0651:1, L0805:1, L0657:1, L0659:1, L0809:1, L0790:1, L0663:1, L0438:1, S0126:1, S0406:1, H0555:1, H0436:1, S0312:1, S0027:1, L0745:1, L0749:1, L0750:1, L0780:1, H0707:1, S0436:1, L0591:1 and S0242:1.
	HWLEV32	873296	295		
	HWLEV32	881710	296		
	HWLEV32	846351	297		
190	HYBAR01	610383	200		AR308:42, AR192:7, AR205:4, AR161:3, AR198:3, AR178:3, AR162:3, AR163:3, AR193:3, AR216:3, AR169:3, AR176:3, AR270:3, AR089:3, AR246:3, AR269:3, AR204:3, AR291:3, AR039:2, AR164:2, AR254:2, AR215:2, AR053:2, AR257:2, AR171:2, AR195:2, AR271:2, AR201:2, AR277:2, AR266:2, AR060:2, AR316:2, AR173:2, AR282:2, AR275:2, AR262:2, AR264:2, AR213:2, AR288:2, AR104:2, AR272:1, AR182:1, AR225:1, AR183:1, AR166:1, AR311:1, AR294:1, AR165:1, AR299:1, AR283:1, AR229:1, AR181:1, AR312:1, AR217:1 H0041:1, L0471:1 and L0766:1.
191	HYBBE75	834784	201		AR215:6, AR252:4, AR162:4, AR161:4, AR163:4, AR183:3, AR309:3, AR165:3, AR164:3, AR176:3, AR235:3, AR166:3, AR270:3, AR204:3, AR245:3, AR192:3, AR216:3, AR193:2, AR242:2, AR257:2, AR277:2, AR196:2, AR089:2, AR201:2, AR250:2, AR266:2, AR313:2, AR182:2, AR291:2, AR255:2, AR233:2, AR060:2, AR282:2, AR225:2, AR197:2, AR214:2, AR239:2, AR247:2, AR294:2, AR185:2, AR293:2, AR268:2, AR285:2, AR177:2, AR213:2, AR287:2, AR178:2, AR237:1, AR174:1, AR230:1, AR267:1, AR316:1, AR240:1, AR181:1, AR096:1, AR228:1, AR290:1, AR286:1, AR232:1, AR296:1, AR262:1, AR189:1, AR061:1, AR221:1, AR289:1, AR226:1, AR179:1, AR238:1, AR236:1, AR295:1, AR300:1, AR210:1 H0041:1
192	HAPSA79	846517	202		AR186:8, AR310:7, AR274:6, AR033:6, AR218:5, AR313:5, AR104:5, AR219:5, AR202:5, AR226:5, AR039:4, AR055:4, AR183:4, AR246:4, AR184:4, AR238:3, AR192:3, AR177:3, AR163:3, AR247:3, AR175:3, AR309:3, AR275:3, AR089:3, AR273:3, AR206:3, AR271:3, AR251:3, AR162:3, AR161:3, AR164:3, AR292:3, AR282:3, AR166:3, AR096:3, AR237:3, AR176:3, AR243:3, AR227:3, AR240:3, AR235:3, AR299:3, AR232:2, AR185:2, AR259:2, AR269:2, AR061:2, AR165:2, AR300:2, AR245:2, AR053:2, AR225:2, AR221:2, AR249:2, AR270:2, AR204:2, AR296:2, AR268:2, AR277:2, AR312:2, AR316:2, AR261:2, AR241:2, AR272:2, AR213:2, AR224:2, AR242:2, AR267:2, AR284:2, AR257:2, AR052:2, AR201:2, AR295:2, AR266:2, AR291:2, AR193:2, AR294:1, AR231:1, AR173:1, AR233:1, AR197:1, AR060:1, AR253:1, AR195:1, AR293:1, AR207:1, AR217:1, AR286:1, AR308:1, AR205:1, AR285:1, AR172:1, AR178:1, AR179:1, AR290:1, AR256:1, AR181:1, AR216:1, AR228:1, AR214:1, AR198:1, AR212:1, AR229:1, AR244:1, AR171:1, AR168:1, AR182:1, AR311:1 L0731:12, L0747:9, H0651:5, L0759:5, H0644:4, H0013:3, L0748:3, L0439:3, L0779:3, H0575:2, H0052:2, H0327:2, H0050:2, H0083:2, L0769:2, L0662:2, L0438:2, H0539:2, L0743:2, L0750:2, L0588:2, H0716:1, L0002:1, L0443:1, S0001:1, S0360:1, H0645:1, H0411:1, H0587:1, H0333:1, H0486:1, S0010:1, S0050:1, H0051:1, H0428:1, H0553:1, H0032:1, L0455:1, S0036:1, H0038:1, H0412:1, H0413:1, H0100:1, T0042:1, L0770:1, L0637:1, L0766:1, L0649:1, L0774:1, L0776:1, L0655:1, L0659:1, L0783:1, L0529:1, L5623:1, L0790:1, L0791:1, H0520:1, H0519:1, H0593:1, H0689:1, H0670:1,

				H0672:1, H0696:1, S3014:1, L0741:1, L0744:1, L0757:1, L0608:1 and S0398:1.	
	HAPSA79	887467	298		
	HAPSA79	878627	299		

Table 1C summarizes additional polynucleotides encompassed by the invention (including cDNA clones related to the sequences (Clone ID:), contig sequences (contig identifier (Contig ID:) contig nucleotide sequence identifiers (SEQ ID NO:X)), and genomic sequences (SEQ ID NO:B). The first column provides a unique clone identifier, "Clone ID:", for a cDNA clone related to each contig sequence. The second column provides the sequence identifier, "SEQ ID NO:X", for each contig sequence. The third column provides a unique contig identifier, "Contig ID:" for each contig sequence. The fourth column, provides a BAC identifier "BAC ID NO:A" for the BAC clone referenced in the corresponding row of the table. The fifth column provides the nucleotide sequence identifier, "SEQ ID NO:B" for a fragment of the BAC clone identified in column four of the corresponding row of the table. The sixth column, "Exon From-To", provides the location (i.e., nucleotide position numbers) within the polynucleotide sequence of SEQ ID NO:B which delineate certain polynucleotides of the invention that are also exemplary members of polynucleotide sequences that encode polypeptides of the invention (e.g., polypeptides containing amino acid sequences encoded by the polynucleotide sequences delineated in column six, and fragments and variants thereof).

Table 1C

cDNA Clone ID	SEQ ID NO:X	CONTIG ID:	BAC ID: A	SEQ ID NO:B	EXON From-To
HCE3G69	34	728432	AC068946	589	1-108 1186-1324 1746-1835 2138-2284 2448-2545 2718-2861 3091-5889
HCE3G69	34	728432	AC068946	590	1-191
HCE3G69	34	728432	AC068946	591	1-686
HDPWN93	53	992925	AC004590	592	1-276 489-591 866-988 1106-1281 1323-1444 1632-1799 1866-2016 2109-2313 2634-3205 3360-3472 3528-3744 3820-5006 6580-6919 7076-7276

					8057-8153 8318-8680
HDPWN93	53	992925	AC021491	593	1-275 488-590 865-987 1105-1280 1322-1443 1631-1798 1865-2015 2108-2312 2633-3204 3359-3471 3527-3743 3819-5005 6579-6918 7075-7275 8054-8150 8315-8677
HDPWN93	53	992925	AC004590	594	1-303 727-1252 5721-5846
HDPWN93	53	992925	AC021491	595	1-303 727-1253 5723-5848
HE8QV67	56	1050076	AL133410	596	1-765 4403-4496 4696-4813 5112-5584 5780-5830 5850-7766 7774-8284 8479-8902 8986-9110 9305-9481 9658-9944 9998-10106 10202-12718 12797-12886 12974-13063 13259-14645 14680-14941 15625-15714 15825-15895 15965-16114 16204-16772
HE8QV67	56	1050076	AL133410	597	1-85 1082-1951 2761-3118
HE8QV67	56	1050076	AL133410	598	1-26 28-267 828-3952 4173-4837

					4930-6955 7105-7230 7451-7655 7842-7947 8245-8329 8599-8756 8855-8940 9219-9356 9728-9861 10190-10231
HEBBN36	59	486120	AC005180	599	1-341 704-1559 1704-3089 3146-4166 4768-4871 5384-5485 5535-6182 6595-7328
HEBBN36	59	486120	AC002557	600	1-1387
HEBBN36	59	486120	AC002557	601	1-856
HEBBN36	59	486120	AC002557	602	1-971
HFIIZ70	66	1043350	AC005005	603	1-368 1579-2971
HFIIZ70	66	1043350	AC005005	604	1-484 517-1142 2842-3176 3376-3493 3575-3740 3873-4227 4728-4935 5074-5351 5446-5564 5772-5960 7287-7627 7721-8097 8218-9325 12098-12161 12780-13266 13482-13666 13748-13817 14445-14519 14595-14928 15658-15754 15848-15923 16016-16112 16512-16660 21313-21448 21710-21870 21899-22470 22634-22787 23169-23307
HFOXA73	69	850699	AC005866	605	1-523

HFOXA73	69	850699	AC007618	606	1-522
HHENK42	79	493724	AC023105	607	1-192 355-585 1654-1995 3493-3802 3827-4082 5266-5952 6109-6292 6819-6947 7118-8308 8602-8887 9337-9517 10052-10284 10616-11071
HHENK42	79	493724	AC023105	608	1-286
HHENK42	79	493724	AC023105	609	1-754
HKACM93	92	1352383	AL158848	610	1-431 4227-4418 6907-7028 12393-12788 13026-13171 14505-14634 14659-14701 15118-15405 16371-16568 17704-17888 18408-18580 18868-19021 19843-20023 21731-21911 23724-25211
HKACM93	92	1352383	AL158848	611	1-2833 2990-3408 3932-5958 5960-6045 6428-6501
HLJBJ61	101	1019012	AC010422	612	1-326 1552-2084 2162-2261 2300-2427 4485-5868 5948-6362 7914-8017 8569-8681 8765-8875 8968-9037 9284-9499 9742-9910 10837-11187 11271-11321 11554-11707 11783-12766

					12866-13225 13256-13827 14284-14367 14890-15090
HLJBJ61	101	1019012	AC018761	613	1-326 1176-1284 1552-2084 2162-2261 2300-2426 4485-5868 5948-6362 8569-8681 8765-8875 8968-9037 9284-9499 9742-9910 10317-10501 10837-11187 11271-11321 11554-11707 11783-12766 12866-13225 13256-13827 14284-14367 14890-15090
HLJBJ61	101	1019012	AC010422	614	1-315 2004-2289 2650-2741 3554-3830
HLJBJ61	101	1019012	AC010422	615	1-202 938-1047 1219-1395 1758-1956 2907-3429 3792-3935 5366-5485 6391-6688 6899-7269 7890-8316 8400-8524 8607-8682 8824-8999 9190-9302 9691-9796 10106-10177 10571-11051 11164-11490 12565-12696 13364-13501 13964-14592 14740-15540 15610-15798

					15947-16642 16717-16832 16968-17408 17521-17612 18331-18579 19120-19303 19358-19514 19599-19702 20003-20245
HLJB61	101	1019012	AC018761	616	1-202 938-1047 1219-1395 1758-1956 2907-3429 3792-3935 5366-5485 6391-6688 6899-7269 7591-7711 7890-8316 8400-8524 8607-8682 8749-9073 9190-9302 9691-9796
HLJB61	101	1019012	AC018761	617	1-82 128-293 1178-1447 1986-2278 2457-2711 3543-3844
HNGOI12	125	1041375	AC003675	618	1-2128
HNGOI12	125	1041375	AC001228	619	1-2129
HNGOI12	125	1041375	AC013791	620	1-2132
HPJBK12	144	1011467	AC022033	621	1-2649
HPJBK12	144	1011467	AC013541	622	1-2649
HPJBK12	144	1011467	AC022033	623	1-190
HPJBK12	144	1011467	AC013541	624	1-190
HPJEX20	145	1352420	AL080251	625	1-1821
HPJEX20	145	1352420	AL139283	626	1-1821
HPJEX20	145	1352420	AL080251	627	1-313
HPJEX20	145	1352420	AL139283	628	1-313
HPWAY46	149	1001560	AC019036	629	1-1399
HPWAY46	149	1001560	AC067828	630	1-1399
HPWAY46	149	1001560	AC019036	631	1-788
HPWAY46	149	1001560	AC067828	632	1-788
HSYAZ50	165	1027673	AC007378	633	1-2471
HSYAZ50	165	1027673	AC073041	634	1-2471
HSYAZ50	165	1027673	AC007378	635	1-467
HSYAZ50	165	1027673	AC073041	636	1-467
HTHBG43	177	919911	AL139257	637	1-36 130-201

					330-753 1823-2214 2331-2440 2728-2834 2920-3028 3370-3514 4153-5236 5877-6744 6813-7124 8441-9280 9527-9953 10394-10536 10945-11362 11763-11843 12653-12953 13970-14183 14223-14726 15929-16299 16328-16751 17791-18093 18095-18712 18754-24628 24879-25426
HTHBG43	177	919911	AL139257	638	1-286
HTLIV19	181	1046341	AC055750	639	1-964
HTLIV19	181	1046341	AC027463	640	1-964
HTLIV19	181	1046341	AC055750	641	1-236
HTLIV19	181	1046341	AC027463	642	1-236

Tables 1D: The polynucleotides or polypeptides, or agonists or antagonists of the present invention can be used in assays to test for one or more biological activities. If these polynucleotides and polypeptides do exhibit activity in a particular assay, it is likely that these molecules may be involved in the diseases associated with the biological activity. Thus, the polynucleotides or polypeptides, or agonists or antagonists could be used to treat the associated disease.

The present invention encompasses methods of detecting, preventing, diagnosing, prognosticating, treating, and/or ameliorating a disease or disorder. In preferred embodiments, the present invention encompasses a method of treating an allergic and/or asthmatic disease or disorder comprising administering to a patient in which such detection, treatment, prevention, and/or amelioration is desired a protein, nucleic acid, or antibody of the invention (or fragment or variant thereof) in an amount effective to detect, prevent, diagnose, prognosticate, treat, and/or ameliorate the allergic and/or asthmatic disease or disorder.

In another embodiment, the present invention also encompasses methods of detecting, preventing, diagnosing, prognosticating, treating, and/or ameliorating an allergic and/or asthmatic

disease or disorder; comprising administering to a patient combinations of the proteins, nucleic acids, or antibodies of the invention (or fragments or variants thereof), sharing similar indications as shown in the corresponding rows in Column 3 of Table 1D.

5 Table 1D provides information related to biological activities for polynucleotides and polypeptides of the invention (including antibodies, agonists, and/or antagonists thereof). Table 1D also provides information related to assays which may be used to test polynucleotides and polypeptides of the invention (including antibodies, agonists, and/or antagonists thereof) for the corresponding biological activities. The first column ("Gene No.") provides the gene number in the application for each clone identifier. The second column ("cDNA Clone ID:") provides the
10 unique clone identifier for each clone as previously described and indicated in Table 1A through Table 1D. The third column ("AA SEQ ID NO:Y") indicates the Sequence Listing SEQ ID Number for polypeptide sequences encoded by the corresponding cDNA clones (also as indicated in Tables 1A, Table 1B, and Table 2). The fourth column ("Biological Activity") indicates a biological activity corresponding to the indicated polypeptides (or polynucleotides encoding said
15 polypeptides). The fifth column ("Exemplary Activity Assay") further describes the corresponding biological activity and also provides information pertaining to the various types of assays that may be performed to test, demonstrate, or quantify the corresponding biological activity.

Table 1D describes the use of, inter alia, FMAT technology for testing or demonstrating various biological activities. Fluorometric microvolume assay technology (FMAT) is a
20 fluorescence-based system that provides a means to perform nonradioactive cell- and bead-based assays to detect activation of cell signal transduction pathways. This technology was designed specifically for ligand binding and immunological assays. Using this technology, fluorescent cells or beads at the bottom of the well are detected as localized areas of concentrated fluorescence using a data processing system. Unbound fluorphore comprising the background signal is ignored,
25 allowing for a wide variety of homogeneous assays. FMAT technology may be used for peptide ligand binding assays, immunofluorescence, apoptosis, cytotoxicity, and bead-based immunocapture assays. *See*, Miraglia S et. al., "Homogeneous cell and bead based assays for highthroughput screening using fluorometric microvolume assay technology," Journal of Biomolecular Screening; 4:193-204 (1999). In particular, FMAT technology may be used to test,
30 confirm, and/or identify the ability of polypeptides (including polypeptide fragments and variants) to activate signal transduction pathways. For example, FMAT technology may be used to test, confirm, and/or identify the ability of polypeptides to upregulate production of immunomodulatory proteins (such as, for example, interleukins, GM-CSF, Rantes, and Tumor Necrosis factors, as well as other cellular regulators (e.g. insulin)).

35

Table 1D also describes the use of kinase assays for testing, demonstrating, or quantifying biological activity. In this regard, the phosphorylation and de-phosphorylation of specific amino acid residues (e.g. Tyrosine, Serine, Threonine) on cell-signal transduction proteins provides a fast, reversible means for activation and de-activation of cellular signal transduction pathways.

5 Moreover, cell signal transduction via phosphorylation/de-phosphorylation is crucial to the regulation of a wide variety of cellular processes (e.g. proliferation, differentiation, migration, apoptosis, etc.). Accordingly, kinase assays provide a powerful tool useful for testing, confirming, and/or identifying polypeptides (including polypeptide fragments and variants) that mediate cell

10 signal transduction events via protein phosphorylation. See e.g., Forrer, P., Tamaskovic R., and Jaussi, R. "Enzyme-Linked Immunosorbent Assay for Measurement of JNK, ERK, and p38 Kinase Activities" *Biol. Chem.* 379(8-9): 1101-1110 (1998).

Table 1D

Gene No.	cDNA Clone ID	AA SEQ ID NO: Y	Biological Activity	Exemplary Activity Assay	Preferred Indication
1	H2MAC30	305	Activation of transcription through serum response element in immune cells (such as T-cells).	Assays for the activation of transcription through the Serum Response Element (SRE) are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate the serum response factors and modulate the expression of genes involved in growth. Exemplary assays for transcription through the SRE that may be used or routinely modified to test SRE activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-	A preferred embodiment of the invention includes a method for inhibiting (e.g., reducing) TNF alpha production. An alternative preferred embodiment of the invention includes a method for stimulating (e.g., increasing) TNF alpha production. Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"), Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated

			<p>368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); and Black et al., Virus Genes 12(2):105-117 (1997), the content of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary mouse T cells that may be used according to these assays include the CTLL cell line, which is an IL-2 dependent suspension culture of T cells with cytotoxic activity.</p>	<p>immune response, and suppressing a T cell-mediated immune response. Additional highly preferred indications include inflammation and inflammatory disorders, and treating joint damage in patients with rheumatoid arthritis. An additional highly preferred indication is sepsis. Highly preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Additionally, highly preferred indications include neoplasms and cancers, such as, for example, leukemia, lymphoma, melanoma, glioma (e.g., malignant glioma), solid tumors, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for</p>
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					<p>example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, cardiac reperfusion injury, and asthma and allergy. An additional preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease").</p>
1	H2MAC30	305	<p>Activation of JNK Signaling Pathway in immune cells (such as eosinophils).</p>	<p>Kinase assay. JNK kinase assays for signal transduction that regulate cell proliferation, activation, or apoptosis are well known in the art and may</p>	<p>Highly preferred indications include asthma, allergy, hypersensitivity reactions, inflammation, and inflammatory disorders.</p>

				<p>be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and apoptosis. Exemplary assays for JNK kinase activity that may be used or routinely modified to test JNK kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. Exemplary cells that may be</p>	<p>Additional highly preferred indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below). Highly preferred indications also include boosting or inhibiting immune cell proliferation. Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include boosting an eosinophil-mediated immune response, and suppressing an eosinophil-mediated immune response.</p>
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				<p>used according to these assays include eosinophils.</p> <p>Eosinophils are important in the late stage of allergic reactions; they are recruited to tissues and mediate the inflammatory response of late stage allergic reaction.</p> <p>Moreover, exemplary assays that may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate signal transduction, cell proliferation, activation, or apoptosis in eosinophils include assays disclosed and/or cited in: Zhang JP, et al., "Role of caspases in dexamethasone-induced apoptosis and activation of c-Jun NH2-terminal kinase and p38 mitogen-activated protein kinase in human eosinophils" Clin Exp Immunol; Oct;122(1):20-7 (2000); Hebestreit H, et al., "Disruption of fas receptor</p>	
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				signaling by nitric oxide in eosinophils" J Exp Med; Feb 2;187(3):415-25 (1998); J Allergy Clin Immunol 1999 Sep;104(3 Pt 1):565-74; and, Sousa AR, et al., "In vivo resistance to corticosteroids in bronchial asthma is associated with enhanced phosphorylation of JUN N-terminal kinase and failure of prednisolone to inhibit JUN N-terminal kinase phosphorylation" J Allergy Clin Immunol; Sep;104(3 Pt 1):565-74 (1999); the contents of each of which are herein incorporated by reference in its entirety.	
2	H6EDF66	306	Inhibition of squalene synthetase gene transcription.	Reporter Assay: construct contains regulatory and coding sequence of squalene synthetase, the first specific enzyme in the cholesterol biosynthetic pathway. See Jiang, et al., J. Biol. Chem. 268:12818-12824(1993), the contents of which are herein incorporated by reference in its entirety. Cells were treated	

				with SID supernatants, and SEAP activity was measured after 72 hours. HepG2 is a human hepatocellular carcinoma cell line (ATCC HB-8065). See Knowles et al., Science. 209:497-9 (1980), the contents of which are herein incorporated by reference in its entirety.	
2	H6EDF66	306	Activation or inhibition of transcription through NFkB response element in immune cells (such as basophils).	This reporter assay measures activation or inhibition of the NFkB signaling pathway in Ku812 human basophil cell line. Assays for the activation or inhibition of transcription through the NFkB response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFkB transcription factors and modulate expression of immunomodulatory genes. NFkB is important in the pathogenesis of asthma.	

				<p>Exemplary assays for transcription through the NFkB response element that may be used or routinely modified to test NFkB-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Marone et al, Int Arch Allergy Immunol 114(3):207-17 (1997), the contents of each of which are herein incorporated by reference in its entirety. Cells were pretreated with SID supernatants or controls for 15-18 hours, and then 10 ng/mL of TNF was added to stimulate the NFkB reporter. SEAP activity was measured after 48 hours. Basophils that may be used according to these assays are publicly available (e.g.,</p>	
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				through the ATCC). Exemplary human basophil cell lines that may be used according to these assays include Ku812, originally established from a patient with chronic myelogenous leukemia. It is an immature prebasophilic cell line that can be induced to differentiate into mature basophils. See, Kishi et al., Leuk Res. 9:381-390 (1985); Blom et al., Eur J Immunol. 22:2025-32 (1992), where the contents of each are herein incorporated by reference in its entirety.	
2	H6EDF66	306	Activation of Transcription	Assays for activation of transcription are well-known in the art and may be used and routinely modified to assess ability of polypeptides of the invention to inhibit or activate transcription. An example of such an assay follows: Cells were pretreated with SID supernatants or controls for 15-18 hours. SEAP activity was measured after 48 hours. LS174T is an epithelial colon	

				adenocarcinoma cell line. Its tumorigenicity in nude mice make cell line LS174T a model for studies on the mechanism of synthesis and secretion of specific tumoral markers in colon cancer. See, Patan et al., Circ Res, 89(8):732-39 (2001), the contents of which are herein incorporated by reference in its entirety.	
2	H6EDF66	306	Hexosaminidase in RBL-2H3		
2	H6EDF66	306	Activation of transcription through AP1 response element in immune cells (such as T-cells).	Assays for the activation of transcription through the AP1 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate growth and other cell functions. Exemplary assays for transcription through the AP1 response element that may be used or routinely modified to test AP1-response element activity of polypeptides of the	Preferred indications include neoplastic diseases (e.g., as described below under "Hyperproliferative Disorders"), blood disorders (e.g., as described below under "Immune Activity", "Cardiovascular Disorders", and/or "Blood-Related Disorders"), and infection (e.g., an infectious disease as described below under "Infectious Disease"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple

				<p>invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1988); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Rellahan et al., J Biol Chem 272(49):30806-30811 (1997); Chang et al., Mol Cell Biol 18(9):4986-4993 (1998); and Fraser et al., Eur J Immunol 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. Human T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is an IL-2 and IL-4 responsive suspension-culture cell line.</p>	<p>sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Additional highly preferred indications include inflammation and inflammatory disorders. Highly preferred indications also include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, leukemia, lymphoma, prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver, and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include arthritis, asthma, AIDS, allergy, anemia, pancytopenia, leukopenia, thrombocytopenia,</p>
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					<p>Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, granulomatous disease, inflammatory bowel disease, sepsis, psoriasis, suppression of immune reactions to transplanted organs and tissues, endocarditis, meningitis, and Lyme Disease.</p>
2	H6EDF66	306	<p>Production of IL-10 and activation of T-cells.</p>	<p>Assays for production of IL-10 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell</p>	<p>Highly preferred indications include allergy and asthma. Additional highly preferred indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response.</p>

				<p>proliferation include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing</p>	
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3	H6EDX46	307	<p>Activation of transcription through serum response element in immune cells (such as T-cells).</p>	<p>conditions using peripheral blood lymphocytes isolated from cord blood.</p> <p>Assays for the activation of transcription through the Serum Response Element (SRE) are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate the serum response factors and modulate the expression of genes involved in growth. Exemplary assays for transcription through the SRE that may be used or routinely modified to test SRE activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA</p>	<p>A preferred embodiment of the invention includes a method for inhibiting (e.g., reducing) TNF alpha production. An alternative preferred embodiment of the invention includes a method for stimulating (e.g., increasing) TNF alpha production. Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"), Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated</p>
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				<p>immune response. Additional highly preferred indications include inflammation and inflammatory disorders, and treating joint damage in patients with rheumatoid arthritis. An additional highly preferred indication is sepsis. Highly preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Additionally, highly preferred indications include neoplasms and cancers, such as, for example, leukemia, lymphoma, melanoma, glioma (e.g., malignant glioma), solid tumors, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia.</p>
			<p>85:6342-6346 (1988); and Black et al., Virus Genes 12(2):105-117 (1997), the content of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary mouse T cells that may be used according to these assays include the CTLL cell line, which is an IL-2 dependent suspension culture of T cells with cytotoxic activity.</p>	

				agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL10 secreted from Th2 cells may be	"Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response.
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				<p>measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.</p>	
4	HABAG37	308	<p>Activation of transcription through GAS response element in immune cells (such as T-cells).</p>	<p>Assays for the activation of transcription through the Gamma Interferon Activation Site (GAS) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate STAT transcription factors and modulate gene expression involved in a wide variety of cell functions. Exemplary</p>	<p>Highly preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, for example, leukemia, lymphoma (e.g., T cell lymphoma, Burkitt's lymphoma, non-Hodgkins lymphoma, Hodgkin's disease), melanoma, and prostate, breast, lung, colon, pancreatic,</p>

				<p>assays for transcription through the GAS response element that may be used or routinely modified to test GAS-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Matikainen et al., Blood 93(6):1980-1991 (1999); and Henttinen et al., J Immunol 155(10):4582-4587 (1995), the contents of each of which are herein incorporated by reference in its entirety. Exemplary human T cells, such as the MOLT4 cell line, that may be used according to these assays are publicly available (e.g., through the ATCC).</p>	<p>esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional preferred indications include inflammation and inflammatory disorders. Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"), and infection (e.g., viral infections, tuberculosis,</p>
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					infections associated with chronic granulomatous disease and malignant osteoporosis, and/or an infectious disease as described below under "Infectious Disease"). An additional preferred indication is idiopathic pulmonary fibrosis. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, and asthma and allergy.
4	HABAG37	308	Activation of transcription through NFKB response element in	Assays for the activation of transcription through the NFKB response element are well-known in the art and may	Highly preferred indications include inflammation and inflammatory disorders. Highly preferred indications

			immune cells (such as T-cells).	be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFkB transcription factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription through the NFkB response element that may be used or routinely modified to test NFkB-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Black et al., Virus Gnes 15(2):105-117 (1997); and Fraser et al., 29(3):838-844 (1999), the contents of each of which are herein incorporated by	include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), and immunodeficiencies (e.g., as described below). An additional highly preferred indication is infection (e.g., AIDS, and/or an infectious disease as described below under "Infectious Disease"). Highly preferred indications include neoplastic diseases (e.g., melanoma, leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, for example, melanoma, renal cell carcinoma, leukemia, lymphoma, and prostate,
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				<p>reference in its entirety. Exemplary human T cells, such as the MOL T4, that may be used according to these assays are publicly available (e.g., through the ATCC).</p>	<p>breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications also include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, suppression of immune reactions to transplanted organs, asthma and allergy.</p>
5	HACBD91	309	<p>Activation of transcription through cAMP response element</p>	<p>Assays for the activation of transcription through the cAMP response element are well-known in the art and may</p>	<p>A highly preferred indication is obesity and/or complications associated with obesity. Additional highly preferred</p>

		(CRE) in pre-adipocytes.	<p>be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to increase cAMP, regulate CREB transcription factors, and modulate expression of genes involved in a wide variety of cell functions. For example, a 3T3-L1/CRE reporter assay may be used to identify factors that activate the cAMP signaling pathway. CREB plays a major role in adipogenesis, and is involved in differentiation into adipocytes. CRE contains the binding sequence for the transcription factor CREB (CRE binding protein). Exemplary assays for transcription through the cAMP response element that may be used or routinely modified to test cAMP-response element activity of polypeptides of the invention (including antibodies and</p>	<p>indications include weight loss or alternatively, weight gain. An additional highly preferred indication is diabetes mellitus. An additional highly preferred indication is a complication associated with diabetes (e.g., diabetic retinopathy, diabetic nephropathy, kidney disease (e.g., renal failure, nephropathy and/or other diseases and disorders as described in the "Renal Disorders" section below), diabetic neuropathy, nerve disease and nerve damage (e.g., due to diabetic neuropathy), blood vessel blockage, heart disease, stroke, impotence (e.g., due to diabetic neuropathy or blood vessel blockage), seizures, mental confusion, drowsiness, nonketotic hyperglycemic hyperosmolar coma, cardiovascular disease (e.g., heart disease, atherosclerosis, microvascular disease, hypertension, stroke, and other diseases and disorders as</p>
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				<p>agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Reusch et al., Mol Cell Biol 20(3):1008-1020 (2000); and Klemm et al., J Biol Chem 273:917-923 (1998), the contents of each of which are herein incorporated by reference in its entirety. Pre-adipocytes that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary mouse adipocyte cells that may be used according to these assays include 3T3-L1 cells. 3T3-L1 is an adherent mouse preadipocyte cell line that is a continuous substrain of 3T3 fibroblast cells developed through clonal isolation and undergo a pre-adipocyte to</p>	<p>described in the "Cardiovascular Disorders" section below), dyslipidemia, endocrine disorders (as described in the "Endocrine Disorders" section below), neuropathy, vision impairment (e.g., diabetic retinopathy and blindness), ulcers and impaired wound healing, and infection (e.g., infectious diseases and disorders as described in the "Infectious Diseases" section below, especially of the urinary tract and skin), carpal tunnel syndrome and Dupuytren's contracture). Additional highly preferred indications are complications associated with insulin resistance.</p>
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5	HACBD91	309	<p>Activation of transcription through cAMP response element in immune cells (such as T-cells).</p>	<p>Assays for the activation of transcription through the cAMP response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to increase cAMP and regulate CREB transcription factors, and modulate expression of genes involved in a wide variety of cell functions. Exemplary assays for transcription through the cAMP response element that may be used or routinely modified to test cAMP-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol</p>	<p>adipose-like conversion under appropriate differentiation conditions known in the art.</p>	<p>Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"), and infection (e.g., an infectious disease as described below under "Infectious Disease"). Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional preferred indications include inflammation and inflammatory disorders. Highly preferred indications include neoplastic diseases (e.g., leukemia, lymphoma,</p>
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				<p>216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Black et al., Virus Genes 15(2):105-117 (1997); and Belkowski et al., J Immunol 161(2):659-665 (1998), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary mouse T cells that may be used according to these assays include the CTLL cell line, which is a suspension culture of IL-2 dependent cytotoxic T cells.</p>	<p>and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, for example, leukemia, lymphoma (e.g., T cell lymphoma, Burkitt's lymphoma, non-Hodgkins lymphoma, Hodgkin's disease), melanoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis,</p>
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					suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, and asthma and allergy.
5	HACBD91	309	Production of IL-6	<p>IL-6 FMAT. IL-6 is produced by T cells and has strong effects on B cells. IL-6 participates in IL-4 induced IgE production and increases IgA production (IgA plays a role in mucosal immunity). IL-6 induces cytotoxic T cells. Deregulated expression of IL-6 has been linked to autoimmune disease, plasmacytomas, myelomas, and chronic hyperproliferative diseases. Assays for immunomodulatory and differentiation factor and proteins produced by a large variety of cells where the expression level is strongly regulated by cytokines, growth factors, and hormones are well known in the art and may be used or routinely modified to assess the ability of</p>	<p>A highly preferred embodiment of the invention includes a method for stimulating (e.g., increasing) IL-6 production. An alternative highly preferred embodiment of the invention includes a method for inhibiting (e.g., reducing) IL-6 production. A highly preferred indication is the stimulation or enhancement of mucosal immunity. Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"), and infection (e.g., as described below under "Infectious Disease"). Highly preferred indications include autoimmune diseases (e.g.,</p>

				<p>polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation and differentiation and modulate T cell proliferation and function. Exemplary assays that test for immunomodulatory proteins evaluate the production of cytokines, such as IL-6, and the stimulation and upregulation of T cell proliferation and functional activities. Such assays that may be used or routinely modified to test immunomodulatory and differentiation activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204(1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); and Verhasselt et al., J Immunol 158:2919-2925</p>	<p>rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Highly preferred indications also include boosting a B cell-mediated immune response and alternatively suppressing a B cell-mediated immune response. Highly preferred indications include inflammation and inflammatory disorders. Additional highly preferred indications include asthma and allergy. Highly preferred indications include neoplastic diseases (e.g., myeloma, plasmacytoma, leukemia, lymphoma, melanoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, myeloma, plasmacytoma, leukemia, lymphoma, melanoma, and</p>
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				<p>(1997), the contents of each of which are herein incorporated by reference in its entirety. Human dendritic cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human dendritic cells are antigen presenting cells in suspension culture, which, when activated by antigen and/or cytokines, initiate and upregulate T cell proliferation and functional activities.</p> <p>prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, and Lyme Disease. An additional preferred indication is infection (e.g., an infectious disease as described below under "Infectious</p>
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5	HACBD91	309	Regulation of transcription of Malic Enzyme in adipocytes	Assays for the regulation of transcription of Malic Enzyme are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate transcription of Malic Enzyme, a key enzyme in lipogenesis. Malic enzyme is involved in lipogenesis and its expression is stimulated by insulin. ME promoter contains two direct repeat (DR1)- like elements MEp and ME _d identified as putative PPAR response elements. ME promoter may also responds to API and other transcription factors. Exemplary assays that may be used or routinely modified to test for regulation of transcription of Malic Enzyme (in adipocytes) by polypeptides of the invention (including antibodies and agonists or antagonists of the	Disease"). A highly preferred indication is diabetes mellitus. An additional highly preferred indication is a complication associated with diabetes (e.g., diabetic retinopathy, diabetic nephropathy, kidney disease (e.g., renal failure, nephropathy and/or other diseases and disorders as described in the "Renal Disorders" section below), diabetic neuropathy, nerve disease and nerve damage (e.g., due to diabetic neuropathy), blood vessel blockage, heart disease, stroke, impotence (e.g., due to diabetic neuropathy or blood vessel blockage), seizures, mental confusion, drowsiness, nonketotic hyperglycemic-hyperosmolar coma, cardiovascular disease (e.g., heart disease, atherosclerosis, microvascular disease, hypertension, stroke, and other diseases and disorders as described in the
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				<p>invention) include assays disclosed in: Streeper, R.S., et al., Mol Endocrinol, 12(11):1778-91 (1998); Garcia-Jimenez, C., et al., Mol Endocrinol, 8(10):1361-9 (1994); Barroso, I., et al., J Biol Chem, 274(25):17997-8004 (1999); Ijpenberg, A., et al., J Biol Chem, 272(32):20108-20117 (1997); Berger, et al., Gene 66:1-10 (1988); and, Cullen, B., et al., Methods in Enzymol. 216:362-368 (1992), the contents of each of which is herein incorporated by reference in its entirety. Hepatocytes that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary hepatocytes that may be used according to these assays includes the H4IIE rat liver hepatoma cell line.</p>	<p>"Cardiovascular Disorders" section below), dyslipidemia, endocrine disorders (as described in the "Endocrine Disorders" section below), neuropathy, vision impairment (e.g., diabetic retinopathy and blindness), ulcers and impaired wound healing, and infection (e.g., infectious diseases and disorders as described in the "Infectious Diseases" section below, especially of the urinary tract and skin), carpal tunnel syndrome and Dupuytren's contracture). An additional highly preferred indication is obesity and/or complications associated with obesity. Additional highly preferred indications include weight loss or alternatively, weight gain. Additional highly preferred indications are complications associated with insulin resistance.</p>
5	HACBD91	309	Activation of Endothelial Cell p38 or JNK	<p>Kinase assay. JNK and p38 kinase assays for signal transduction that regulate cell</p>	<p>A highly preferred embodiment of the invention includes a method for</p>

			<p>proliferation, activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and apoptosis. Exemplary assays for JNK and p38 kinase activity that may be used or routinely modified to test JNK and p38 kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which</p>	<p>stimulating endothelial cell growth. An alternative highly preferred embodiment of the invention includes a method for inhibiting endothelial cell growth. A highly preferred embodiment of the invention includes a method for stimulating endothelial cell proliferation. An alternative highly preferred embodiment of the invention includes a method for inhibiting endothelial cell proliferation. A highly preferred embodiment of the invention includes a method for stimulating apoptosis of endothelial cells. An alternative highly preferred embodiment of the invention includes a method for inhibiting apoptosis of endothelial cells. A highly preferred embodiment of the invention includes a method for stimulating endothelial cell activation. An alternative highly preferred</p>
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			are herein incorporated by reference in its entirety. Endothelial cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary endothelial cells that may be used according to these assays include human umbilical vein endothelial cells (HUVEC), which are endothelial cells which line venous blood vessels, and are involved in functions that include, but are not limited to, angiogenesis, vascular permeability, vascular tone, and immune cell extravasation.	embodiment of the invention includes a method for inhibiting (e.g., decreasing) the activation of and/or inactivating endothelial cells. A highly preferred embodiment of the invention includes a method for stimulating angiogenesis. An alternative highly preferred embodiment of the invention includes a method for inhibiting angiogenesis. A highly preferred embodiment of the invention includes a method for reducing cardiac hypertrophy. An alternative highly preferred embodiment of the invention includes a method for inducing cardiac hypertrophy. Highly preferred indications include neoplastic diseases (e.g., as described below under "Hyperproliferative Disorders"), and disorders of the cardiovascular system (e.g., heart disease, congestive heart failure, hypertension, aortic stenosis,
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					<p>cardiomyopathy, valvular regurgitation, left ventricular dysfunction, atherosclerosis and atherosclerotic vascular disease, diabetic nephropathy, intracardiac shunt, cardiac hypertrophy, myocardial infarction, chronic hemodynamic overload, and/or as described below under “Cardiovascular Disorders”).</p> <p>Highly preferred indications include cardiovascular, endothelial and/or angiogenic disorders (e.g., systemic disorders that affect vessels such as diabetes mellitus, as well as diseases of the vessels themselves, such as of the arteries, capillaries, veins and/or lymphatics). Highly preferred are indications that stimulate angiogenesis and/or cardiovascularization. Highly preferred are indications that inhibit angiogenesis and/or cardiovascularization.</p> <p>Highly preferred indications include antiangiogenic activity to treat solid tumors,</p>
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					<p>leukemias, and Kaposi's sarcoma, and retinal disorders. Highly preferred indications include neoplasms and cancer, such as, Kaposi's sarcoma, hemangioma (capillary and cavernous), glomus tumors, telangiectasia, bacillary angiomatosis, hemangioendothelioma, angiosarcoma, haemangiopericytoma, lymphangioma, lymphangiosarcoma. Highly preferred indications also include cancers such as, prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver, and urinary cancer. Preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Highly preferred indications also include arterial disease, such as, atherosclerosis, hypertension, coronary artery disease, inflammatory</p>
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					<p>vasculitides, Reynaud's disease and Reynaud's phenomenon, aneurysms, restenosis; venous and lymphatic disorders such as thrombophlebitis, lymphangitis, and lymphedema; and other vascular disorders such as peripheral vascular disease, and cancer. Highly preferred indications also include trauma such as wounds, burns, and injured tissue (e.g., vascular injury such as, injury resulting from balloon angioplasty, and atherosclerotic lesions), implant fixation, scarring, ischemia reperfusion injury, rheumatoid arthritis, cerebrovascular disease, renal diseases such as acute renal failure, and osteoporosis. Additional highly preferred indications include stroke, graft rejection, diabetic or other retinopathies, thrombotic and coagulative disorders, vasculitis, lymph</p>
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					<p>angiogenesis, sexual disorders, age-related macular degeneration, and treatment/prevention of endometriosis and related conditions. Additional highly preferred indications include fibromas, heart disease, cardiac arrest, heart valve disease, and vascular disease.</p> <p>Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders").</p> <p>Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Additional preferred indications include inflammation and inflammatory disorders (such as acute and chronic inflammatory diseases, e.g., inflammatory bowel disease</p>
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					and Crohn's disease), and pain management.
5	HACBD91	309	Activation of transcription through CD28 response element in immune cells (such as T-cells).	<p>Assays for the activation of transcription through the CD28 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate IL-2 expression in T cells. Exemplary assays for transcription through the CD28 response element that may be used or routinely modified to test CD28-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); McGuire and Iacobelli, J Immunol 159(3):1319-1327</p>	<p>A highly preferred embodiment of the invention includes a method for stimulating T cell proliferation. An alternative highly preferred embodiment of the invention includes a method for inhibiting T cell proliferation. A highly preferred embodiment of the invention includes a method for activating T cells. An alternative highly preferred embodiment of the invention includes a method for inhibiting the activation of and/or inactivating T cells. A highly preferred embodiment of the invention includes a method for stimulating (e.g., increasing) IL-2 production. An alternative highly preferred embodiment of the invention includes a method for inhibiting (e.g., reducing) IL-2 production. Additional highly preferred indications include</p>

				<p>(1997); Parra et al., J Immunol 166(4):2437-2443 (2001); and Butscher et al., J Biol Chem 3(1):552-560 (1998), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the JURKAT cell line, which is a suspension culture of leukemia cells that produce IL-2 when stimulated.</p>	<p>inflammation and inflammatory disorders. Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. An additional highly preferred indication includes infection (e.g., AIDS, and/or as described below under "Infectious Disease").</p> <p>Highly preferred indications include neoplastic diseases (e.g., melanoma, renal cell carcinoma, leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, for example, melanoma (e.g., metastatic melanoma), renal</p>
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					<p>cell carcinoma (e.g., metastatic renal cell carcinoma), leukemia, lymphoma (e.g., T cell lymphoma), and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. A highly preferred indication is infection (e.g., tuberculosis, infections associated with granulomatous disease, and osteoporosis, and/or an infectious disease as described below under "Infectious Disease"). A highly preferred indication is AIDS.</p> <p>Additional highly preferred indications include suppression of immune reactions to transplanted organs and/or tissues, uveitis, psoriasis, and tropical spastic paraparesis. Preferred indications include blood disorders (e.g., as</p>
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					described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Preferred indications also include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, asthma and allergy.
5	HACBD91	309	Activation of transcription through NFAT response element in immune cells (such as natural killer cells).	Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate	Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as

				<p>NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Aramburu et al., J Exp Med 182(3):801-810 (1995); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Fraser et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J Biol Chem 268(19):14285-14293 (1993), the contents of each of which are herein incorporated by</p>	<p>described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional highly preferred indications include inflammation and inflammatory disorders. An additional highly preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease"). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Preferred indications include neoplasms and cancers, such as, for example, leukemia, lymphoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and</p>
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				reference in its entirety. NK cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human NK cells that may be used according to these assays include the NK-YT cell line, which is a human natural killer cell line with cytolytic and cytotoxic activity.	pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications also include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, asthma and allergy.
5	HACBD91	309	Activation of transcription through serum response element in immune cells (such as natural killer cells).	Assays for the activation of transcription through the Serum Response Element (SRE) are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or	A preferred embodiment of the invention includes a method for inhibiting (e.g., reducing) TNF alpha production. An alternative highly preferred embodiment of the invention includes a method for stimulating (e.g., increasing) TNF alpha

			<p>antagonists of the invention) to regulate serum response factors and modulate the expression of genes involved in growth and upregulate the function of growth-related genes in many cell types. Exemplary assays for transcription through the SRE that may be used or routinely modified to test SRE activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Benson et al., J Immunol 153(9):3862-3873 (1994); and Black et al., Virus Genes 12(2):105-117 (1997), the content of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g.,</p>	<p>production. Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"), Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional highly preferred indications include inflammation and inflammatory disorders, and treating joint damage in patients with rheumatoid arthritis. An additional highly preferred indication is sepsis. Highly preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below</p>
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				<p>through the ATCC). Exemplary T cells that may be used according to these assays include the NK-YT cell line, which is a human natural killer cell line with cytolytic and cytotoxic activity.</p> <p>under "Hyperproliferative Disorders"). Additionally, highly preferred indications include neoplasms and cancers, such as, for example, leukemia, lymphoma, melanoma, glioma (e.g., malignant glioma), solid tumors, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, neutropenia, neutrophilia, psoriasis, suppression of immune</p>
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					reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, cardiac reperfusion injury, and asthma and allergy. An additional preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease").
5	HACBD91	309	Activation of transcription through API response element in immune cells (such as T-cells).	Assays for the activation of transcription through the API response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate growth and other cell functions. Exemplary assays for transcription through the API response element that may be used or routinely modified to test API-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of	Preferred indications include neoplastic diseases (e.g., as described below under "Hyperproliferative Disorders"), blood disorders (e.g., as described below under "Immune Activity", "Cardiovascular Disorders", and/or "Blood-Related Disorders"), and infection (e.g., an infectious disease as described below under "Infectious Disease"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and

				<p>the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1988); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Rellahan et al., J Biol Chem 272(49):30806-30811 (1997); Chang et al., Mol Cell Biol 18(9):4986-4993 (1998); and Fraser et al., Eur J Immunol 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. Human T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is an IL-2 and IL-4 responsive suspension-culture cell line.</p>	<p>immunodeficiencies (e.g., as described below). Additional highly preferred indications include inflammation and inflammatory disorders. Highly preferred indications also include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, leukemia, lymphoma, prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver, and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include arthritis, asthma, AIDS, allergy, anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL),</p>
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					<p>plasmacytomas, multiple myeloma, Burkitt's lymphoma, granulomatous disease, inflammatory bowel disease, sepsis, psoriasis, suppression of immune reactions to transplanted organs and tissues, endocarditis, meningitis, and Lyme Disease.</p>
5	HACBD91	309	<p>Activation of transcription through CD28 response element in immune cells (such as T-cells).</p>	<p>Assays for the activation of transcription through the CD28 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate IL-2 expression in T cells. Exemplary assays for transcription through the CD28 response element that may be used or routinely modified to test CD28-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene</p>	<p>A highly preferred embodiment of the invention includes a method for stimulating T cell proliferation. An alternative highly preferred embodiment of the invention includes a method for inhibiting T cell proliferation. A highly preferred embodiment of the invention includes a method for activating T cells. An alternative highly preferred embodiment of the invention includes a method for inhibiting the activation of and/or inactivating T cells. A highly preferred embodiment of the invention includes a method for stimulating (e.g., increasing)</p>

				<p>66:1-10 (1998); Cullen and Malm, <i>Methods in Enzymol</i> 216:362-368 (1992); Henthorn et al., <i>Proc Natl Acad Sci USA</i> 85:6342-6346 (1988); McGuire and Iacobelli, <i>J Immunol</i> 159(3):1319-1327 (1997); Parra et al., <i>J Immunol</i> 166(4):2437-2443 (2001); and Butscher et al., <i>J Biol Chem</i> 3(1):552-560 (1998), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.</p>	<p>IL-2 production. An alternative highly preferred embodiment of the invention includes a method for inhibiting (e.g., reducing) IL-2 production. Additional highly preferred indications include inflammation and inflammatory disorders. Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Highly preferred indications include neoplastic diseases (e.g., melanoma, renal cell carcinoma, leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, for</p>
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					<p>example, melanoma (e.g., metastatic melanoma), renal cell carcinoma (e.g., metastatic renal cell carcinoma), leukemia, lymphoma (e.g., T cell lymphoma), and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. A highly preferred indication includes infection (e.g., AIDS, tuberculosis, infections associated with granulomatous disease, and osteoporosis, and/or as described below under "Infectious Disease"). A highly preferred indication is AIDS. Additional highly preferred indications include suppression of immune reactions to transplanted organs and/or tissues, uveitis, psoriasis, and tropical spastic paraparesis. Preferred</p>
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					indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Preferred indications also include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, asthma and allergy.
5	HACBD91	309	Activation of transcription through NFAT response element in immune cells (such as T-cells).	Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies	Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis,

				<p>and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., <i>Gene</i> 66:1-10 (1998); Cullen and Malm, <i>Methods in Enzymol</i> 216:362-368 (1992); Henthorn et al., <i>Proc Natl Acad Sci USA</i> 85:6342-6346 (1988); Serfling et al., <i>Biochim Biophys Acta</i> 1498(1):1-18 (2000); De Boer et al., <i>Int J Biochem Cell Biol</i> 31(10):1221-1236 (1999); Fraser et al., <i>Eur J Immunol</i> 29(3):838-844 (1999); and Yeseen et al., <i>J Biol Chem</i> 268(19):14285-14293 (1993),</p>	<p>systemic lupus erythematosus, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional highly preferred indications include inflammation and inflammatory disorders. An additional highly preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease"). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Preferred indications include neoplasms and cancers, such as, for example, leukemia, lymphoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred</p>
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				<p>the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.</p>	<p>indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications also include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, asthma and allergy.</p>
5	HACBD91	309	<p>Activation of transcription through NFkB response element in immune cells (such as T-cells).</p>	<p>Assays for the activation of transcription through the NFkB response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention</p>	<p>Highly preferred indications include inflammation and inflammatory disorders. Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-</p>

				<p>(including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription through the NFKB response element that may be used or routinely modified to test NFKB-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Black et al., Virus Gnes 15(2):105-117 (1997); and Fraser et al., 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are</p>	<p>Related Disorders", and/or "Cardiovascular Disorders"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), and immunodeficiencies (e.g., as described below). An additional highly preferred indication is infection (e.g., AIDS, and/or an infectious disease as described below under "Infectious Disease"). Highly preferred indications include neoplastic diseases (e.g., melanoma, leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, melanoma, renal cell carcinoma, leukemia, lymphoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other</p>
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				publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.	preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications also include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, suppression of immune reactions to transplanted organs, asthma and allergy.
5	HACBD91	309	Activation of transcription through STAT6 response element in immune cells (such as T-cells).	Assays for the activation of transcription through the Signal Transducers and Activators of Transcription (STAT6) response element are well-known in the art and may	<p>A highly preferred indication is allergy.</p> <p>Another highly preferred indication is asthma.</p> <p>Additional highly preferred indications include</p>

			<p>be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate STAT6 transcription factors and modulate the expression of multiple genes. Exemplary assays for transcription through the STAT6 response element that may be used or routinely modified to test STAT6 response element activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Georas et al., Blood 92(12):4529-4538 (1998); Moffatt et al., Transplantation 69(7):1521-1523 (2000); Curiel et al., Eur J Immunol 27(8):1982-1987</p>	<p>inflammation and inflammatory disorders. Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, melanoma, and/or as described below under "Hyperproliferative Disorders"). Preferred indications include neoplasms and cancers, such as, leukemia, lymphoma, melanoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred</p>
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				<p>(1997); and Masuda et al., J Biol Chem 275(38):29331-29337 (2000), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.</p>	<p>indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, and Lyme Disease. An additional preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease").</p>
6	HACCI17	310	Activation of Adipocyte ERK Signaling Pathway	Kinase assay. Kinase assays, for example an Elk-1 kinase assay, for ERK signal	<p>A highly preferred embodiment of the invention includes a method for</p>

			transduction that regulate cell proliferation or differentiation are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and differentiation. Exemplary assays for ERK kinase activity that may be used or routinely modified to test ERK kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Le Marchand-Brustel Y, Exp Clin Endocrinol Diabetes 107(2):126-132 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol	stimulating adipocyte proliferation. An alternative highly preferred embodiment of the invention includes a method for inhibiting adipocyte proliferation. A highly preferred embodiment of the invention includes a method for stimulating adipocyte differentiation. An alternative highly preferred embodiment of the invention includes a method for inhibiting adipocyte differentiation. A highly preferred embodiment of the invention includes a method for stimulating (e.g., increasing) adipocyte activation. An alternative highly preferred embodiment of the invention includes a method for inhibiting the activation of (e.g., decreasing) and/or inactivating adipocytes. Highly preferred indications include endocrine disorders (e.g., as described below under "Endocrine Disorders"). Highly preferred indications
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				<p>Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety.</p> <p>Mouse adipocyte cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary mouse adipocyte cells that may be used according to these assays include 3T3-L1 cells. 3T3-L1 is an adherent mouse preadipocyte cell line that is a continuous substrain of 3T3 fibroblast cells developed through clonal isolation and undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation conditions known in the art.</p>	<p>also include neoplastic diseases (e.g., lipomas, liposarcomas, and/or as described below under "Hyperproliferative Disorders"). Preferred indications include blood disorders (e.g., hypertension, congestive heart failure, blood vessel blockage, heart disease, stroke, impotence and/or as described below under "Immune Activity", "Cardiovascular Disorders", and/or "Blood-Related Disorders"), immune disorders (e.g., as described below under "Immune Activity"), neural disorders (e.g., as described below under "Neural Activity and Neurological Diseases"), and infection (e.g., as described below under "Infectious Disease").</p> <p>A highly preferred indication is diabetes mellitus. An additional highly preferred indication is a complication associated with diabetes (e.g., diabetic retinopathy, diabetic</p>
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					<p>nephropathy, kidney disease (e.g., renal failure, nephropathy and/or other diseases and disorders as described in the "Renal Disorders" section below), diabetic neuropathy, nerve disease and nerve damage (e.g., due to diabetic neuropathy), blood vessel blockage, heart disease, stroke, impotence (e.g., due to diabetic neuropathy or blood vessel blockage), seizures, mental confusion, drowsiness, nonketotic hyperglycemic hyperosmolar coma, cardiovascular disease (e.g., heart disease, atherosclerosis, microvascular disease, hypertension, stroke, and other diseases and disorders as described in the "Cardiovascular Disorders" section below), dyslipidemia, endocrine disorders (as described in the "Endocrine Disorders" section below), neuropathy, vision impairment (e.g., diabetic retinopathy and</p>
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					<p>blindness), ulcers and impaired wound healing, infection (e.g., infectious diseases and disorders as described in the "Infectious Diseases" section below (particularly of the urinary tract and skin). An additional highly preferred indication is obesity and/or complications associated with obesity. Additional highly preferred indications include weight loss or alternatively, weight gain. Additional highly preferred indications are complications associated with insulin resistance.</p> <p>Additional highly preferred indications are disorders of the musculoskeletal systems including myopathies, muscular dystrophy, and/or as described herein.</p> <p>Additional highly preferred indications include, hypertension, coronary artery disease, dyslipidemia, gallstones, osteoarthritis, degenerative arthritis, eating disorders, fibrosis, cachexia,</p>
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					and kidney diseases or disorders. Preferred indications include neoplasms and cancer, such as, lymphoma, leukemia and breast, colon, and kidney cancer. Additional preferred indications include melanoma, prostate, lung, pancreatic, esophageal, stomach, brain, liver, and urinary cancer. Highly preferred indications include lipomas and liposarcomas. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia.
6	HACCI17	310	Production of IL-8 by immune cells (such as the human EOL-1 eosinophil cells)	Assay that measures the production of the chemokine interleukin-8 (IL-8) from immune cells (such as the EOL-1 human eosinophil cell line) are well known in the art (for example, measurement of IL-8 production by FMT) and may be used or routinely modified to assess the ability of polypeptides of the	Highly preferred indications include eosinophilia, asthma, allergy, hypersensitivity reactions, inflammation, and inflammatory disorders. Additional highly preferred indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"),

			<p>invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit. Eosinophils are a type of immune cell important in allergic responses; they are recruited to tissues and mediate the inflammatory response of late stage allergic reaction. IL8 is a strong immunomodulator and may have a potential proinflammatory role in immunological diseases and disorders (such as allergy and asthma).</p>	<p>autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below). Highly preferred indications also include boosting or inhibiting immune cell proliferation. Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include boosting an eosinophil-mediated immune response, and suppressing an eosinophil-mediated immune response.</p>
6	HACCI17	310	<p>Activation of transcription through GATA-3 response element in immune cells (such as mast cells).</p>	<p>This reporter assay measures activation of the GATA-3 signaling pathway in HMC-1 human mast cell line. Activation of GATA-3 in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription</p> <p>Highly preferred indications include allergy, asthma, and rhinitis. Additional preferred indications include infection (e.g., an infectious disease as described below under "Infectious Disease"), and inflammation and inflammatory disorders.</p>

				<p>through the GATA3 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3 transcription factors and modulate expression of mast cell genes important for immune response development. Exemplary assays for transcription through the GATA3 response element that may be used or routinely modified to test GATA3-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Flavell et al., Cold Spring Harb Symp Quant Biol 64:563-571 (1999);</p>	<p>Preferred indications also include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, melanoma, prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver, and urinary tract cancers and/or as described below under "Hyperproliferative Disorders"). Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include</p>
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				<p>Rodriguez-Palmero et al., Eur J Immunol 29(12):3914-3924 (1999); Zheng and Flavell, Cell 89(4):587-596 (1997); and Henderson et al., Mol Cell Biol 14(6):4286-4294 (1994), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC).</p> <p>Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.</p>	<p>anemia, pancytopenia, leukopenia, thrombocytopenia, leukemias, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, and Lyme Disease.</p>
6	HACCI17	310	<p>Activation of transcription through NFAT response element in immune cells (such as mast cells).</p>	<p>This reporter assay measures activation of the NFAT signaling pathway in HMC-1 human mast cell line. Activation of NFAT in mast cells has been linked to cytokine and chemokine</p>	<p>Highly preferred indications include allergy, asthma, and rhinitis. Additional preferred indications include infection (e.g., an infectious disease as described below under "Infectious Disease"), and</p>

				<p>production. Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn</p>	<p>inflammation and inflammatory disorders. Preferred indications also include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, melanoma, prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver, and urinary tract cancers and/or as described below under "Hyperproliferative Disorders"). Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia,</p>
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				<p>et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Ali et al., J Immunol 165(12):7215-7223 (2000); Hutchinson and McCloskey, J Biol Chem 270(27):16333-16338 (1995), and Turner et al., J Exp Med 188:527-537 (1998), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.</p>	<p>metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, leukemias, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, and Lyme Disease.</p>
6	HACCI17	310	IgG in Human B cells SAC	IL-5 FMAT. Assays for	A highly preferred
	HACCI17	310	Production of IL-5		

6				<p>immunomodulatory proteins secreted by TH2 cells, mast cells, basophils, and eosinophils that stimulate eosinophil function and B cell Ig production and promote polarization of CD4+ cells into TH2 cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation, stimulate immune cell function, modulate B cell Ig production, modulate immune cell polarization, and/or mediate humoral or cell-mediated immunity.</p> <p>Exemplary assays that test for immunomodulatory proteins evaluate the production of cytokines, such as IL-5, and the stimulation of eosinophil function and B cell Ig production. Such assays that may be used or routinely modified to test</p>	<p>embodiment of the invention includes a method for inhibiting (e.g., reducing) IL-5 production. An alternative highly preferred embodiment of the invention includes a method for stimulating (e.g., increasing) IL-5 production. A highly preferred embodiment of the invention includes a method for stimulating (e.g., increasing) immunoglobulin production. An alternative highly preferred embodiment of the invention includes a method for inhibiting (e.g., decreasing) immunoglobulin production. A highly preferred indication includes allergy. A highly preferred indication includes asthma. A highly preferred indication includes rhinitis. An additional highly preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease"), and inflammation and inflammatory disorders. Preferred indications include</p>
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				<p>immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204 (1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); Ohshima et al., Blood 92(9):3338-3345 (1998); Jung et al., Eur J Immunol 25(8):2413-2416 (1995); Mori et al., J Allergy Clin Immunol 106(1 Pt 2):558-564 (2000); and Koning et al., Cytokine 9(6):427-436 (1997), the contents of each of which are herein incorporated by reference in its entirety.</p> <p>Human T cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human T cells are primary human lymphocytes that mature in the thymus and express a T cell receptor and</p>	<p>blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, melanoma, and/or as described below under "Hyperproliferative Disorders"). Preferred indications include neoplasms and cancers, such as, leukemia, lymphoma, melanoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia,</p>
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				CD3, CD4, or CD8. These cells mediate humoral or cell-mediated immunity and may be preactivated to enhance responsiveness to immunomodulatory factors.	metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, leukemias, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, and Lyme Disease.
6	HACII17	310	Production of ICAM in endothelial cells (such as human umbilical vein endothelial cells (HUVEC))	Endothelial cells, which are cells that line blood vessels, and are involved in functions that include, but are not limited to, angiogenesis, vascular permeability, vascular tone, and immune cell extravasation. Exemplary endothelial cells that may be used in ICAM production assays include human umbilical vein	Highly preferred indications include inflammation (acute and chronic), restenosis, atherosclerosis, asthma and allergy. Highly preferred indications include inflammation and inflammatory disorders, immunological disorders, neoplastic disorders (e.g. cancer/tumorigenesis), and

				<p>endothelial cells (HUVEC), and are available from commercial sources. The expression of ICAM (CD54),^a integral membrane protein, can be upregulated by cytokines or other factors, and ICAM expression is important in mediating immune and endothelial cell interactions leading to immune and inflammatory responses. Assays for measuring expression of ICAM-1 are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate ICAM-1 expression. Exemplary assays that may be used or routinely modified to measure ICAM-1 expression include assays disclosed in: Rolfe BE, et al., <i>Atherosclerosis</i>, 149(1):99-110 (2000); Panettieri RA Jr, et al., <i>J Immunol</i>, 154(5):2358-2365 (1995); and, Grunstein MM, et</p>	<p>cardiovascular disorders (such as described below under "Immune Activity", "Blood-Related Disorders", "Hyperproliferative Disorders" and/or "Cardiovascular Disorders"). Highly preferred indications include neoplasms and cancers such as, for example, leukemia, lymphoma, melanoma, renal cell carcinoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia.</p>
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				al., Am J Physiol Lung Cell Mol Physiol, 278(6):L1154-L1163 (2000), the contents of each of which is herein incorporated by reference in its entirety.	
6	HACCI17	310	Production of IL-8 by endothelial cells (such as Human Umbilical Cord Endothelial Cells).	Assays measuring production of IL-8 are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate production and/or secretion of IL-8. For example, FMAT may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate production and/or secretion of IL-8 from endothelial cells (such as human umbilical vein endothelial cells (HUVEC)). HUVECs are endothelial cells which line venous blood vessels, and are involved in	Highly preferred indications include immunological and inflammatory disorders (e.g., such as allergy, asthma, leukemia, etc. and as described below under "Immune Activity", and "Blood-Related Disorders"). Highly preferred indications also include autoimmune disorders (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), neoplastic disorders (e.g., organ cancers such as lung, liver, colon cancer, and/or as described below under "Hyperproliferative Disorders"), and cardiovascular disorders (e.g., such as described below under "Cardiovascular Disorders"). Preferred indications include

				<p>functions that include, but are not limited to, angiogenesis, vascular permeability, vascular tone, and immune cell extravasation. Endothelial cells play a pivotal role in the initiation and perpetuation of inflammation and secretion of IL-8 may play an important role in recruitment and activation of immune cells such as neutrophils, macrophages, and lymphocytes.</p>	<p>thrombosis, bacteremia and sepsis syndrome and consequent complications (such as acute respiratory distress syndrome and systemic ischemia-reperfusion resulting from septic shock), restnosis and atherosclerosis.</p>
6	HACCI17	310	<p>Production of RANTES in endothelial cells (such as human umbilical vein endothelial cells (HUVEC))</p>	<p>RANTES FMAT. Assays for immunomodulatory proteins that induce chemotaxis of T cells, monocytes, and eosinophils are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation, induce chemotaxis, and/or mediate humoral or cell-mediated immunity. Exemplary assays that test for</p>	

				<p>immunomodulatory proteins evaluate the production of cytokines, such as RANTES, and the induction of chemotactic responses in immune cells. Such assays that may be used or routinely modified to test immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204 (1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); Cocchi et al., Science 270(5243):1811-1815 (1995); and Robinson et al., Clin Exp Immunol 101(3):398-407 (1995), the contents of each of which are herein incorporated by reference in its entirety. Endothelial cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary endothelial cells</p>	
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				that may be used according to these assays include human umbilical vein endothelial cells (HUVEC), which are endothelial cells which line venous blood vessels, and are involved in functions that include, but are not limited to, angiogenesis, vascular permeability, vascular tone, and immune cell extravasation.	
6	HACCI17	310	Production of VCAM in endothelial cells (such as human umbilical vein endothelial cells (HUVEC))	Assays for measuring expression of VCAM are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate VCAM expression. For example, FMAT may be used to measure the upregulation of cell surface VCAM-1 expression in endothelial cells. Endothelial cells are cells that line blood vessels, and are involved in functions that include, but are not limited to, angiogenesis, vascular permeability, vascular	Highly preferred indications include inflammation (acute and chronic), restnosis, atherosclerosis, asthma and allergy. Highly preferred indications include inflammation and inflammatory disorders, immunological disorders, neoplastic disorders (e.g. cancer/tumorigenesis), and cardiovascular disorders (such as described below under "Immune Activity", "Blood-Related Disorders", "Hyperproliferative Disorders" and/or "Cardiovascular Disorders"). Highly preferred indications include neoplasms

				tone, and immune cell extravasation. Exemplary endothelial cells that may be used according to these assays include human umbilical vein endothelial cells (HUVEC), which are available from commercial sources. The expression of VCAM (CD106), a membrane-associated protein, can be upregulated by cytokines or other factors, and contributes to the extravasation of lymphocytes, leucocytes and other immune cells from blood vessels; thus VCAM expression plays a role in promoting immune and inflammatory responses.	and cancers such as, for example, leukemia, lymphoma, melanoma, renal cell carcinoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia.
7	HAGAM64	311	Regulation of apoptosis of immune cells (such as mast cells).	Caspase Apoptosis. Assays for caspase apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate caspase protease-mediated apoptosis in	Preferred embodiments of the invention include using polypeptides of the invention (or antibodies, agonists, or antagonists thereof) in detection, diagnosis, prevention, and/or treatment of asthma, allergy, hypersensitivity and inflammation.

				<p>immune cells (such as, for example, in mast cells). Mast cells are found in connective and mucosal tissues throughout the body, and their activation via immunoglobulin E - antigen, promoted by T helper cell type 2 cytokines, is an important component of allergic disease. Dysregulation of mast cell apoptosis may play a role in allergic disease and mast cell tumor survival. Exemplary assays for caspase apoptosis that may be used or routinely modified to test caspase apoptosis activity induced by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in: Masuda A, et al., J Biol Chem, 276(28):26107-26113 (2001); Yeatman CF 2nd, et al., J Exp Med, 192(8):1093-1103 (2000); Lee et al., FEBS Lett 485(2-3): 122-126 (2000); Nor et al., J Vasc Res 37(3): 209-218 (2000); and Karsan and</p>
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				<p>Harlan, J Atheroscler Thromb 3(2): 75-80 (1996); the contents of each of which are herein incorporated by reference in its entirety.</p> <p>Immune cells that may be used according to these assays are publicly available (e.g., through commercial sources). Exemplary immune cells that may be used according to these assays include mast cells such as the HMC human mast cell line.</p>	
8	HAHDR32	312	<p>Inhibition of squalene synthetase gene transcription.</p>	<p>Reporter Assay: construct contains regulatory and coding sequence of squalene synthetase, the first specific enzyme in the cholesterol biosynthetic pathway. See Jiang, et al., J. Biol. Chem. 268:12818-12824(1993), the contents of which are herein incorporated by reference in its entirety. Cells were treated with SID supernatants, and SEAP activity was measured after 72 hours. HepG2 is a human hepatocellular carcinoma cell line (ATCC</p>	

8	HAHDR32	312	<p>Activation of transcription through NFKB response element in immune cells (such as T-cells).</p>	<p>HB-8065). See Knowles et al., Science. 209:497-9 (1980), the contents of which are herein incorporated by reference in its entirety.</p> <p>Assays for the activation of transcription through the NFKB response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription through the NFKB response element that may be used or routinely modified to test NFKB-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and</p>	<p>Highly preferred indications include inflammatory and inflammatory disorders.</p> <p>Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders").</p> <p>Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), and immunodeficiencies (e.g., as described below). An additional highly preferred indication is infection (e.g., AIDS, and/or an infectious disease as described below under "Infectious Disease").</p> <p>Highly preferred indications include neoplastic diseases (e.g., melanoma, leukemia,</p>
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				<p>lymphoma, and/or as described below under “Hyperproliferative Disorders”). Highly preferred indications include neoplasms and cancers, such as, for example, melanoma, renal cell carcinoma, leukemia, lymphoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications also include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin’s disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt’s lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, hemophilia, hypercoagulation,</p>
				<p>Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Black et al., Virus Gnes 15(2):105-117 (1997); and Fraser et al., 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. Exemplary human T cells, such as the MOLT4, that may be used according to these assays are publicly available (e.g., through the ATCC).</p>

					diabetes mellitus, endocarditis, meningitis, Lyme Disease, suppression of immune reactions to transplanted organs, asthma and allergy.
9	HAIBO71	313	Endothelial Cell Apoptosis	<p>Caspase Apoptosis. Assays for caspase apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote caspase protease-mediated apoptosis. Induction of apoptosis in endothelial cells supporting the vasculature of tumors is associated with tumor regression due to loss of tumor blood supply. Exemplary assays for caspase apoptosis that may be used or routinely modified to test caspase apoptosis activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Lee et al., FEBS</p>	<p>A highly preferred embodiment of the invention includes a method for stimulating endothelial cell growth. An alternative highly preferred embodiment of the invention includes a method for inhibiting endothelial cell growth. A highly preferred embodiment of the invention includes a method for stimulating endothelial cell proliferation. An alternative highly preferred embodiment of the invention includes a method for inhibiting endothelial cell proliferation. A highly preferred embodiment of the invention includes a method for stimulating apoptosis of endothelial cells. An alternative highly preferred embodiment of the invention includes a method for</p>

				<p>inhibiting (e.g., decreasing) apoptosis of endothelial cells. A highly preferred embodiment of the invention includes a method for stimulating angiogenesis. An alternative highly preferred embodiment of the invention includes a method for inhibiting angiogenesis. A highly preferred embodiment of the invention includes a method for reducing cardiac hypertrophy. An alternative highly preferred embodiment of the invention includes a method for inducing cardiac hypertrophy. Highly preferred indications include neoplastic diseases (e.g., as described below under "Hyperproliferative Disorders"), and disorders of the cardiovascular system (e.g., heart disease, congestive heart failure, hypertension, aortic stenosis, cardiomyopathy, valvular regurgitation, left ventricular dysfunction, atherosclerosis</p>
			<p>Lett 485(2-3): 122-126 (2000); Nor et al., J Vasc Res 37(3): 209-218 (2000); and Karsan and Harlan, J Atheroscler Thromb 3(2): 75-80 (1996); the contents of each of which are herein incorporated by reference in its entirety. Endothelial cells that may be used according to these assays are publicly available (e.g., through commercial sources). Exemplary endothelial cells that may be used according to these assays include bovine aortic endothelial cells (bAEC), which are an example of endothelial cells which line blood vessels and are involved in functions that include, but are not limited to, angiogenesis, vascular permeability, vascular tone, and immune cell extravasation.</p>	

					<p>and atherosclerotic vascular disease, diabetic nephropathy, intracardiac shunt, cardiac hypertrophy, myocardial infarction, chronic hemodynamic overload, and/or as described below under “Cardiovascular Disorders”).</p> <p>Highly preferred indications include cardiovascular, endothelial and/or angiogenic disorders (e.g., systemic disorders that affect vessels such as diabetes mellitus, as well as diseases of the vessels themselves, such as of the arteries, capillaries, veins and/or lymphatics). Highly preferred are indications that stimulate angiogenesis and/or cardiovascularization. Highly preferred are indications that inhibit angiogenesis and/or cardiovascularization.</p> <p>Highly preferred indications include antiangiogenic activity to treat solid tumors, leukemias, and Kaposi's sarcoma, and retinal disorders. Highly preferred indications</p>
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					<p>include neoplasms and cancer, such as, Kaposi's sarcoma, hemangioma (capillary and cavernous), glomus tumors, telangiectasia, bacillary angiomatosis, hemangioendothelioma, angiosarcoma, haemangiopericytoma, lymphangioma, lymphangiosarcoma. Highly preferred indications also include cancers such as, prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver, and urinary cancer. Preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Highly preferred indications also include arterial disease, such as, atherosclerosis, hypertension, coronary artery disease, inflammatory vasculitides, Reynaud's disease and Reynaud's phenomenon, aneurysms,</p>
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					<p>restenosis; venous and lymphatic disorders such as thrombophlebitis, lymphangitis, and lymphedema; and other vascular disorders such as peripheral vascular disease, and cancer. Highly preferred indications also include trauma such as wounds, burns, and injured tissue (e.g., vascular injury such as, injury resulting from balloon angioplasty, and atherosclerotic lesions), implant fixation, scarring, ischemia reperfusion injury, rheumatoid arthritis, cerebrovascular disease, renal diseases such as acute renal failure, and osteoporosis. Additional highly preferred indications include stroke, graft rejection, diabetic or other retinopathies, thrombotic and coagulative disorders, vasculitis, lymph angiogenesis, sexual disorders, age-related macular degeneration, and treatment</p>
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					<p>/prevention of endometriosis and related conditions.</p> <p>Additional highly preferred indications include fibromas, heart disease, cardiac arrest, heart valve disease, and vascular disease.</p> <p>Preferred indications include blood disorders (e.g., as described below under “Immune Activity”, “Blood-Related Disorders”, and/or “Cardiovascular Disorders”).</p> <p>Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Additional preferred indications include inflammation and inflammatory disorders (such as acute and chronic inflammatory diseases, e.g., inflammatory bowel disease and Crohn’s disease), and pain management.</p>
HAIBO71	313	Activation of	Assays for the activation of	Highly preferred indications	

9	transcription through NFAT response element in immune cells (such as natural killer cells).	transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988);	include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional highly preferred indications include inflammation and inflammatory disorders. An additional highly preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease"). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative
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			<p>Aramburu et al., J Exp Med 182(3):801-810 (1995); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Fraser et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J Biol Chem 268(19):14285-14293 (1993), the contents of each of which are herein incorporated by reference in its entirety. NK cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human NK cells that may be used according to these assays include the NK-YT cell line, which is a human natural killer cell line with cytolytic and cytotoxic activity.</p>	<p>Disorders"). Preferred indications include neoplasms and cancers, such as, for example, leukemia, lymphoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications also include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease,</p>
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					asthma and allergy.
9	HAIBO71	313	SEAP in NK16/STAT6		
10	HAF57	314	Regulation of apoptosis of immune cells (such as mast cells).	<p>Caspase Apoptosis. Assays for caspase apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate caspase protease-mediated apoptosis in immune cells (such as, for example, in mast cells). Mast cells are found in connective and mucosal tissues throughout the body, and their activation via immunoglobulin E - antigen, promoted by T helper cell type 2 cytokines, is an important component of allergic disease. Dysregulation of mast cell apoptosis may play a role in allergic disease and mast cell tumor survival. Exemplary assays for caspase apoptosis that may be used or routinely modified to test caspase apoptosis activity</p>	<p>Preferred embodiments of the invention include using polypeptides of the invention (or antibodies, agonists, or antagonists thereof) in detection, diagnosis, prevention, and/or treatment of asthma, allergy, hypersensitivity and inflammation.</p>

				induced by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in: Masuda A, et al., J Biol Chem, 276(28):26107-26113 (2001); Yeatman CF 2nd, et al., J Exp Med, 192(8):1093-1103 (2000); Lee et al., FEBS Lett 485(2-3): 122-126 (2000); Nor et al., J Vasc Res 37(3): 209-218 (2000); and Karsan and Harlan, J Atheroscler Thromb 3(2): 75-80 (1996); the contents of each of which are herein incorporated by reference in its entirety. Immune cells that may be used according to these assays are publicly available (e.g., through commercial sources). Exemplary immune cells that may be used according to these assays include mast cells such as the HMC human mast cell line.	
				Kinase assay. JNK kinase assays for signal transduction that regulate cell proliferation,	A highly preferred embodiment of the invention includes a method for
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			<p>activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and apoptosis. Exemplary assays for JNK kinase activity that may be used or routinely modified to test JNK kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by</p>	<p>stimulating endothelial cell growth. An alternative highly preferred embodiment of the invention includes a method for inhibiting endothelial cell growth. A highly preferred embodiment of the invention includes a method for stimulating endothelial cell proliferation. An alternative highly preferred embodiment of the invention includes a method for inhibiting endothelial cell proliferation. A highly preferred embodiment of the invention includes a method for stimulating apoptosis of endothelial cells. An alternative highly preferred embodiment of the invention includes a method for inhibiting apoptosis of endothelial cells. A highly preferred embodiment of the invention includes a method for stimulating endothelial cell activation. An alternative highly preferred embodiment of the invention</p>
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				reference in its entirety. Endothelial cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary endothelial cells that may be used according to these assays include human umbilical vein endothelial cells (HUVEC), which are endothelial cells which line venous blood vessels, and are involved in functions that include, but are not limited to, angiogenesis, vascular permeability, vascular tone, and immune cell extravasation.	includes a method for inhibiting the activation of and/or inactivating endothelial cells. A highly preferred embodiment of the invention includes a method for stimulating angiogenesis. An alternative highly preferred embodiment of the invention includes a method for inhibiting angiogenesis. A highly preferred embodiment of the invention includes a method for reducing cardiac hypertrophy. An alternative highly preferred embodiment of the invention include a method for inducing cardiac hypertrophy. Highly preferred indications include neoplastic diseases (e.g., as described below under “Hyperproliferative Disorders”), and disorders of the cardiovascular system (e.g., heart disease, congestive heart failure, hypertension, aortic stenosis, cardiomyopathy, valvular regurgitation, left ventricular
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					<p>dysfunction, atherosclerosis and atherosclerotic vascular disease, diabetic nephropathy, intracardiac shunt, cardiac hypertrophy, myocardial infarction, chronic hemodynamic overload, and/or as described below under “Cardiovascular Disorders”).</p> <p>Highly preferred indications include cardiovascular, endothelial and/or angiogenic disorders (e.g., systemic disorders that affect vessels such as diabetes mellitus, as well as diseases of the vessels themselves, such as of the arteries, capillaries, veins and/or lymphatics). Highly preferred are indications that stimulate angiogenesis and/or cardiovascularization. Highly preferred are indications that inhibit angiogenesis and/or cardiovascularization.</p> <p>Highly preferred indications include antiangiogenic activity to treat solid tumors, leukemias, and Kaposi's sarcoma, and retinal disorders.</p>
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					phenomenon, aneurysms, restenosis; venous and lymphatic disorders such as thrombophlebitis, lymphangitis, and lymphedema; and other vascular disorders such as peripheral vascular disease, and cancer. Highly preferred indications also include trauma such as wounds, burns, and injured tissue (e.g., vascular injury such as, injury resulting from balloon angioplasty, and atherosclerotic lesions), implant fixation, scarring, ischemia reperfusion injury, rheumatoid arthritis, cerebrovascular disease, renal diseases such as acute renal failure, and osteoporosis. Additional highly preferred indications include stroke, graft rejection, diabetic or other retinopathies, thrombotic and coagulative disorders, vasculitis, lymph angiogenesis, sexual disorders, age-related macular
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					<p>degeneration, and treatment /prevention of endometriosis and related conditions.</p> <p>Additional highly preferred indications include fibromas, heart disease, cardiac arrest, heart valve disease, and vascular disease.</p> <p>Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders").</p> <p>Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Additional preferred indications include inflammation and inflammatory disorders (such as acute and chronic inflammatory diseases, e.g., inflammatory bowel disease and Crohn's disease), and pain management.</p>
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11	HAMFC93	315	Production of IL-13 and activation of T-cells.	Assays for production of IL-13 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-13 and/or activation of T-cells. Exemplary assays for IL-13 production that may be used or routinely modified to test activity of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) include, for example, assays such as disclosed and/or cited in: Grunig, G, et al., "Requirement for IL-13 independently of IL-4 in Experimental asthma" Science;282: 2261-2263 (1998), and Wills-Karp M, et al., "Interleukin-13: central mediator of allergic asthma" Science; 282: 2258-2261 (1998); the contents of each of	Highly preferred indications include allergy and asthma. Additional highly preferred indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response.
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				<p>which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL13, a Th2 type cytokine, is a potent stimulus for mucus production, airway hyper-responsiveness and allergic asthma. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated in in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.</p>	
12	HAPNY86	316	<p>Activation of transcription through STAT6 response element in immune cells (such as T-cells).</p>	<p>Assays for the activation of transcription through the Signal Transducers and Activators of Transcription (STAT6) response element are well-known in the art and may be used or routinely modified to assess the ability of</p>	<p>A highly preferred indication is allergy. Another highly preferred indication is asthma. Additional highly preferred indications include inflammation and inflammatory disorders.</p>

				<p>polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate STAT6 transcription factors and modulate the expression of multiple genes. Exemplary assays for transcription through the STAT6 response element that may be used or routinely modified to test STAT6 response element activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Georas et al., Blood 92(12):4529-4538 (1998); Moffatt et al., Transplantation 69(7):1521-1523 (2000); Curiel et al., Eur J Immunol 27(8):1982-1987 (1997); and Masuda et al., J Biol Chem 275(38):29331-</p>	<p>Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, melanoma, and/or as described below under "Hyperproliferative Disorders"). Preferred indications include neoplasms and cancers, such as, leukemia, lymphoma, melanoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and</p>
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				<p>29337 (2000), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC).</p> <p>Exemplary T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.</p>	<p>pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, and Lyme Disease. An additional preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease").</p>
13	HATDF29	317	<p>Production of IL-10 and activation of T-cells.</p>	<p>Assays for production of IL-10 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of</p>	<p>Highly preferred indications include allergy and asthma. Additional highly preferred indications include immune and hematopoietic disorders</p>

				<p>polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays</p>	<p>(e.g., as described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response.</p>
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				include Th2 cells. IL10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.	
14	HBAFJ33	318	Activation of JNK Signaling Pathway in immune cells (such as eosinophils).	Kinase assay. JNK kinase assays for signal transduction that regulate cell proliferation, activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and apoptosis.	Highly preferred indications include asthma, allergy, hypersensitivity reactions, inflammation, and inflammatory disorders. Additional highly preferred indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic

				<p>Exemplary assays for JNK kinase activity that may be used or routinely modified to test JNK kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. Exemplary cells that may be used according to these assays include eosinophils. Eosinophils are important in the late stage of allergic reactions; they are recruited to tissues and mediate the inflammatory response of late stage allergic reaction.</p>	<p>lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below). Highly preferred indications also include boosting or inhibiting immune cell proliferation. Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include boosting an eosinophil-mediated immune response, and suppressing an eosinophil-mediated immune response.</p>
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					<p>Moreover, exemplary assays that may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate signal transduction, cell proliferation, activation, or apoptosis in eosinophils include assays disclosed and/or cited in: Zhang JP, et al., "Role of caspases in dexamethasone-induced apoptosis and activation of c-Jun NH2-terminal kinase and p38 mitogen-activated protein kinase in human eosinophils" <i>Clin Exp Immunol</i>; Oct;122(1):20-7 (2000); Hebestreit H, et al., "Disruption of fas receptor signaling by nitric oxide in eosinophils" <i>J Exp Med</i>; Feb 2;187(3):415-25 (1998); <i>J Allergy Clin Immunol</i> 1999 Sep;104(3 Pt 1):565-74; and, Sousa AR, et al., "In vivo resistance to corticosteroids in bronchial asthma is associated</p>
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15	HBAFV19	319	Activation of JNK Signaling Pathway in immune cells (such as eosinophils).	with enhanced phosphorylation of JUN N-terminal kinase and failure of prednisolone to inhibit JUN N-terminal kinase phosphorylation" J Allergy Clin Immunol; Sep;104(3 Pt 1):565-74 (1999); the contents of each of which are herein incorporated by reference in its entirety.	Highly preferred indications include asthma, allergy, hypersensitivity reactions, inflammation, and inflammatory disorders. Additional highly preferred indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below). Highly preferred indications also
				Kinase assay. JNK kinase assays for signal transduction that regulate cell proliferation, activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and apoptosis. Exemplary assays for JNK kinase activity that may be used or routinely modified to test JNK kinase-induced activity of polypeptides of the invention (including antibodies	

				<p>and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. Exemplary cells that may be used according to these assays include eosinophils. Eosinophils are important in the late stage of allergic reactions; they are recruited to tissues and mediate the inflammatory response of late stage allergic reaction. Moreover, exemplary assays that may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of</p>	<p>include boosting or inhibiting immune cell proliferation. Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include boosting an eosinophil-mediated immune response, and suppressing an eosinophil-mediated immune response.</p>
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				<p>the invention) to modulate signal transduction, cell proliferation, activation, or apoptosis in eosinophils include assays disclosed and/or cited in: Zhang JP, et al., "Role of caspases in dexamethasone-induced apoptosis and activation of c-Jun NH2-terminal kinase and p38 mitogen-activated protein kinase in human eosinophils" Clin Exp Immunol; Oct;122(1):20-7 (2000); Hebestreit H, et al., "Disruption of fas receptor signaling by nitric oxide in eosinophils" J Exp Med; Feb 2;187(3):415-25 (1998); J Allergy Clin Immunol 1999 Sep;104(3 Pt 1):565-74; and, Sousa AR, et al., "In vivo resistance to corticosteroids in bronchial asthma is associated with enhanced phosphorylation of JUN N-terminal kinase and failure of prednisolone to inhibit JUN N-terminal kinase phosphorylation" J Allergy</p>	
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					Clin Immunol; Sep;104(3 Pt 1):565-74 (1999); the contents of each of which are herein incorporated by reference in its entirety.	
16	HBIBW67	320	Inhibition of squalene synthetase gene transcription.	Reporter Assay: construct contains regulatory and coding sequence of squalene synthetase, the first specific enzyme in the cholesterol biosynthetic pathway. See Jiang, et al., J. Biol. Chem. 268:12818-12824(1993), the contents of which are herein incorporated by reference in its entirety. Cells were treated with SID supernatants, and SEAP activity was measured after 72 hours. HepG2 is a human hepatocellular carcinoma cell line (ATCC HB-8065). See Knowles et al., Science. 209:497-9 (1980), the contents of which are herein incorporated by reference in its entirety.	A highly preferred embodiment of the invention includes a method for inhibiting (e.g., reducing) IL-5	
16	HBIBW67	320	Production of IL-5	IL-5 FMAT. Assays for immunomodulatory proteins secreted by TH2 cells, mast cells, basophils, and		

				<p>eosinophils that stimulate eosinophil function and B cell Ig production and promote polarization of CD4+ cells into TH2 cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation, stimulate immune cell function, modulate B cell Ig production, modulate immune cell polarization, and/or mediate humoral or cell-mediated immunity.</p> <p>Exemplary assays that test for immunomodulatory proteins evaluate the production of cytokines, such as IL-5, and the stimulation of eosinophil function and B cell Ig production. Such assays that may be used or routinely modified to test immunomodulatory activity of polypeptides of the invention (including antibodies and</p>	<p>production. An alternative highly preferred embodiment of the invention includes a method for stimulating (e.g., increasing) IL-5 production. A highly preferred embodiment of the invention includes a method for stimulating (e.g., increasing) immunoglobulin production. An alternative highly preferred embodiment of the invention includes a method for inhibiting (e.g., decreasing) immunoglobulin production. A highly preferred indication includes allergy. A highly preferred indication includes asthma. A highly preferred indication includes rhinitis. An additional highly preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease"), and inflammation and inflammatory disorders. Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-</p>
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				agonists or antagonists of the invention) include the assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204 (1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); Ohshima et al., Blood 92(9):3338-3345 (1998); Jung et al., Eur J Immunol 25(8):2413-2416 (1995); Mori et al., J Allergy Clin Immunol 106(1 Pt 2):558-564 (2000); and Koning et al., Cytokine 9(6):427-436 (1997), the contents of each of which are herein incorporated by reference in its entirety. Human T cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human T cells are primary human lymphocytes that mature in the thymus and express a T cell receptor and CD3, CD4, or CD8. These cells mediate humoral or cell-mediated immunity and may	Related Disorders", and/or "Cardiovascular Disorders"). Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, melanoma, and/or as described below under "Hyperproliferative Disorders"). Preferred indications include neoplasms and cancers, such as, leukemia, lymphoma, melanoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia,
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				be preactivated to enhance responsiveness to immunomodulatory factors.	leukopenia, thrombocytopenia, leukemias, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, and Lyme Disease.
17	HBIMB51	321	Activation of JNK Signaling Pathway in immune cells (such as eosinophils).	Kinase assay. JNK kinase assays for signal transduction that regulate cell proliferation, activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and apoptosis. Exemplary assays for JNK	Highly preferred indications include asthma, allergy, hypersensitivity reactions, inflammation, and inflammatory disorders. Additional highly preferred indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's

				<p>kinase activity that may be used or routinely modified to test JNK kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. Exemplary cells that may be used according to these assays include eosinophils. Eosinophils are important in the late stage of allergic reactions; they are recruited to tissues and mediate the inflammatory response of late stage allergic reaction. Moreover, exemplary assays</p>	<p>disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below). Highly preferred indications also include boosting or inhibiting immune cell proliferation. Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include boosting an eosinophil-mediated immune response, and suppressing an eosinophil-mediated immune response.</p>
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				<p>that may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate signal transduction, cell proliferation, activation, or apoptosis in eosinophils include assays disclosed and/or cited in: Zhang JP, et al., "Role of caspases in dexamethasone-induced apoptosis and activation of c-Jun NH2-terminal kinase and p38 mitogen-activated protein kinase in human eosinophils" Clin Exp Immunol; Oct;122(1):20-7 (2000); Hebestreit H, et al., "Disruption of fas receptor signaling by nitric oxide in eosinophils" J Exp Med; Feb 2;187(3):415-25 (1998); J Allergy Clin Immunol 1999 Sep;104(3 Pt 1):565-74; and, Sousa AR, et al., "In vivo resistance to corticosteroids in bronchial asthma is associated with enhanced</p>	
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				phosphorylation of JUN N-terminal kinase and failure of prednisolone to inhibit JUN N-terminal kinase phosphorylation" J Allergy Clin Immunol; Sep;104(3 Pt 1):565-74 (1999); the contents of each of which are herein incorporated by reference in its entirety.	
17	HBIMB51	321	Inhibition of squalene synthetase gene transcription.	Reporter Assay: construct contains regulatory and coding sequence of squalene synthetase, the first specific enzyme in the cholesterol biosynthetic pathway. See Jiang, et al., J. Biol. Chem. 268:12818-12824(1993), the contents of which are herein incorporated by reference in its entirety. Cells were treated with SID supernatants, and SEAP activity was measured after 72 hours. HepG2 is a human hepatocellular carcinoma cell line (ATCC HB-8065). See Knowles et al., Science. 209:497-9 (1980), the contents of which are herein incorporated by reference in its	

18	HBJID05	322	Activation or inhibition of transcription through NFkB response element in immune cells (such as basophils).	entirety. This reporter assay measures activation or inhibition of the NFkB signaling pathway in Ku812 human basophil cell line. Assays for the activation or inhibition of transcription through the NFkB response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFkB transcription factors and modulate expression of immunomodulatory genes. NFkB is important in the pathogenesis of asthma. Exemplary assays for transcription through the NFkB response element that may be used or routinely modified to test NFkB-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the	
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				<p>invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Marone et al, Int Arch Allergy Immunol 114(3):207-17 (1997), the contents of each of which are herein incorporated by reference in its entirety. Cells were pretreated with SID supernatants or controls for 15-18 hours, and then 10 ng/mL of TNF was added to stimulate the NFkB reporter. SEAP activity was measured after 48 hours. Basophils that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human basophil cell lines that may be used according to these assays include Ku812, originally established from a patient with chronic myelogenous leukemia. It is an immature prebasophilic cell line that can</p>	
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18	HBJID05	322	<p>Production of IL-10 and activation of T-cells.</p>	<p>be induced to differentiate into mature basophils. See, Kishi et al., Leuk Res. 9:381-390 (1985); Blom et al., Eur J Immunol. 22:2025-32 (1992), where the contents of each are herein incorporated by reference in its entirety.</p> <p>Assays for production of IL-10 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in:</p>	<p>Highly preferred indications include allergy and asthma. Additional highly preferred indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response.</p>
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				<p>Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.</p>
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19	HBJU28	323	<p>Production of IL-13 and activation of T-cells.</p>	<p>Assays for production of IL-13 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-13 and/or activation of T-cells. Exemplary assays for IL-13 production that may be used or routinely modified to test activity of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) include, for example, assays such as disclosed and/or cited in: Grunig, G, et al., "Requirement for IL-13 independently of IL-4 in Experimental asthma" Science; 282: 2261-2263 (1998), and Wills-Karp M, et al., "Interleukin-13: central mediator of allergic asthma" Science; 282: 2258-2261 (1998); the contents of each of</p>	<p>Highly preferred indications include allergy and asthma. Additional highly preferred indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response.</p>
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20	HBJLH40	324	<p>Activation of transcription through STAT6 response element in immune cells (such as T-cells).</p>	<p>Assays for the activation of transcription through the Signal Transducers and Activators of Transcription (STAT6) response element are well-known in the art and may be used or routinely modified to assess the ability of</p>	<p>which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL13, a Th2 type cytokine, is a potent stimulus for mucus production, airway hyper-responsiveness and allergic asthma. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated in in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.</p>	<p>A highly preferred indication is allergy. Another highly preferred indication is asthma. Additional highly preferred indications include inflammation and inflammatory disorders.</p>
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				<p>polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate STAT6 transcription factors and modulate the expression of multiple genes. Exemplary assays for transcription through the STAT6 response element that may be used or routinely modified to test STAT6 response element activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Georas et al., Blood 92(12):4529-4538 (1998); Moffatt et al., Transplantation 69(7):1521-1523 (2000); Curiel et al., Eur J Immunol 27(8):1982-1987 (1997); and Masuda et al., J Biol Chem 275(38):29331-</p>	<p>Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, melanoma, and/or as described below under "Hyperproliferative Disorders"). Preferred indications include neoplasms and cancers, such as, leukemia, lymphoma, melanoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and</p>
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				<p>29337 (2000), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.</p>	<p>pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, and Lyme Disease. An additional preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease").</p>
21	HBXFL29	325	<p>Activation of JNK Signaling Pathway in immune cells (such as eosinophils).</p>	<p>Kinase assay. JNK kinase assays for signal transduction that regulate cell proliferation, activation, or apoptosis are well known in the art and may</p>	<p>Highly preferred indications include asthma, allergy, hypersensitivity reactions, inflammation, and inflammatory disorders.</p>

				<p>be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and apoptosis. Exemplary assays for JNK kinase activity that may be used or routinely modified to test JNK kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. Exemplary cells that may be</p>	<p>Additional highly preferred indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below). Highly preferred indications also include boosting or inhibiting immune cell proliferation. Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include boosting an eosinophil-mediated immune response, and suppressing an eosinophil-mediated immune response.</p>
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				<p>used according to these assays include eosinophils.</p> <p>Eosinophils are important in the late stage of allergic reactions; they are recruited to tissues and mediate the inflammatory response of late stage allergic reaction.</p> <p>Moreover, exemplary assays that may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate signal transduction, cell proliferation, activation, or apoptosis in eosinophils include assays disclosed and/or cited in: Zhang JP, et al., "Role of caspases in dexamethasone-induced apoptosis and activation of c-Jun NH2-terminal kinase and p38 mitogen-activated protein kinase in human eosinophils" Clin Exp Immunol; Oct;122(1):20-7 (2000); Hebestreit H, et al., "Disruption of fas receptor</p>	
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22	HCACU58	326	Activation of transcription through serum response element in immune cells (such as T-cells).	Assays for the activation of transcription through the Serum Response Element (SRE) are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate the serum response	signaling by nitric oxide in eosinophils" J Exp Med; Feb 2;187(3):415-25 (1998); J Allergy Clin Immunol 1999 Sep;104(3 Pt 1):565-74; and, Sousa AR, et al., "In vivo resistance to corticosteroids in bronchial asthma is associated with enhanced phosphorylation of JUN N-terminal kinase and failure of prednisolone to inhibit JUN N-terminal kinase phosphorylation" J Allergy Clin Immunol; Sep;104(3 Pt 1):565-74 (1999); the contents of each of which are herein incorporated by reference in its entirety.	A preferred embodiment of the invention includes a method for inhibiting (e.g., reducing) TNF alpha production. An alternative preferred embodiment of the invention includes a method for stimulating (e.g., increasing) TNF alpha production. Preferred indications include blood
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			<p>factors and modulate the expression of genes involved in growth. Exemplary assays for transcription through the SRE that may be used or routinely modified to test SRE activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); and Black et al., Virus Genes 12(2):105-117 (1997), the content of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary mouse T cells that may be used according to these assays include the CTLL cell line, which is an IL-2</p>	<p>disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"), Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional highly preferred indications include inflammation and inflammatory disorders, and treating joint damage in patients with rheumatoid arthritis. An additional highly preferred indication is sepsis. Highly preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Additionally,</p>
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				<p>highly preferred indications include neoplasms and cancers, such as, for example, leukemia, lymphoma, melanoma, glioma (e.g., malignant glioma), solid tumors, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues,</p>
				<p>dependent suspension culture of T cells with cytotoxic activity.</p>

22	HCACU58	326	<p>Activation of transcription through GATA-3 response element in immune cells (such as mast cells).</p>	<p>This reporter assay measures activation of the GATA-3 signaling pathway in HMC-1 human mast cell line. Activation of GATA-3 in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the GATA3 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3 transcription factors and modulate expression of mast cell genes important for immune response</p>	<p>hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, cardiac reperfusion injury, and asthma and allergy. An additional preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease").</p> <p>Highly preferred indications include allergy, asthma, and rhinitis. Additional preferred indications include infection (e.g., an infectious disease as described below under "Infectious Disease"), and inflammation and inflammatory disorders. Preferred indications also include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and</p>
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				<p>development. Exemplary assays for transcription through the GATA3 response element that may be used or routinely modified to test GATA3-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Flavell et al., Cold Spring Harb Symp Quant Biol 64:563-571 (1999); Rodriguez-Palmero et al., Eur J Immunol 29(12):3914-3924 (1999); Zheng and Flavell, Cell 89(4):587-596 (1997); and Henderson et al., Mol Cell Biol 14(6):4286-4294 (1994), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g.,</p>	<p>immunodeficiencies (e.g., as described below). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, melanoma, prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver, and urinary tract cancers and/or as described below under "Hyperproliferative Disorders"). Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, leukemias, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune</p>
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				through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.	reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, and Lyme Disease.
22	HCACU58	326	Production of ICAM in endothelial cells (such as human umbilical vein endothelial cells (HUVEC))	Endothelial cells, which are cells that line blood vessels, and are involved in functions that include, but are not limited to, angiogenesis, vascular permeability, vascular tone, and immune cell extravasation. Exemplary endothelial cells that may be used in ICAM production assays include human umbilical vein endothelial cells (HUVEC), and are available from commercial sources. The expression of ICAM (CD54), ^a integral membrane protein, can be upregulated by cytokines or other factors, and ICAM expression is important	Highly preferred indications include inflammation (acute and chronic), restnosis, atherosclerosis, asthma and allergy. Highly preferred indications include inflammation and inflammatory disorders, immunological disorders, neoplastic disorders (e.g. cancer/tumorigenesis), and cardiovascular disorders (such as described below under "Immune Activity", "Blood-Related Disorders", "Hyperproliferative Disorders" and/or "Cardiovascular Disorders"). Highly preferred indications include neoplasms

				<p>in mediating immune and endothelial cell interactions leading to immune and inflammatory responses. Assays for measuring expression of ICAM-1 are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate ICAM-1 expression. Exemplary assays that may be used or routinely modified to measure ICAM-1 expression include assays disclosed in: Rolfe BE, et al., Atherosclerosis, 149(1):99-110 (2000); Panettieri RA Jr, et al., J Immunol, 154(5):2358-2365 (1995); and, Grunstein MM, et al., Am J Physiol Lung Cell Mol Physiol, 278(6):L1154-L1163 (2000), the contents of each of which is herein incorporated by reference in its entirety.</p>	<p>and cancers such as, for example, leukemia, lymphoma, melanoma, renal cell carcinoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia.</p>
22	HCACU58	326	Production of IL-10 and activation of T-	Assays for production of IL-10 and activation of T-cells are	Highly preferred indications include allergy and asthma.

			cells.	<p>well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by</p>	<p>Additional highly preferred indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response.</p>
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				reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.	
23	HCDBW86	327	Inhibition of squalene synthetase gene transcription.	Reporter Assay: construct contains regulatory and coding sequence of squalene synthetase, the first specific enzyme in the cholesterol biosynthetic pathway. See Jiang, et al., J. Biol. Chem. 268:12818-12824(1993), the contents of which are herein incorporated by reference in its	

				entirety. Cells were treated with SID supernatants, and SEAP activity was measured after 72 hours. HepG2 is a human hepatocellular carcinoma cell line (ATCC HB-8065). See Knowles et al., Science. 209:497-9 (1980), the contents of which are herein incorporated by reference in its entirety.	
23	HCDBW86	327	Production of IL-5	IL-5 FMAT. Assays for immunomodulatory proteins secreted by TH2 cells, mast cells, basophils, and eosinophils that stimulate eosinophil function and B cell Ig production and promote polarization of CD4+ cells into TH2 cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation, stimulate immune cell function, modulate B cell Ig production, modulate immune	<p>A highly preferred embodiment of the invention includes a method for inhibiting (e.g., reducing) IL-5 production. An alternative highly preferred embodiment of the invention includes a method for stimulating (e.g., increasing) IL-5 production.</p> <p>A highly preferred embodiment of the invention includes a method for stimulating (e.g., increasing) immunoglobulin production. An alternative highly preferred embodiment of the invention includes a method for inhibiting (e.g., decreasing) immunoglobulin production.</p>

				<p>cell polarization, and/or mediate humoral or cell-mediated immunity. Exemplary assays that test for immunomodulatory proteins evaluate the production of cytokines, such as IL-5, and the stimulation of eosinophil function and B cell Ig production. Such assays that may be used or routinely modified to test immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204 (1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); Ohshima et al., Blood 92(9):3338-3345 (1998); Jung et al., Eur J Immunol 25(8):2413-2416 (1995); Mori et al., J Allergy Clin Immunol 106(1 Pt 2):558-564 (2000); and Koning et al., Cytokine 9(6):427-436 (1997), the</p>	<p>A highly preferred indication includes allergy. A highly preferred indication includes asthma. A highly preferred indication includes rhinitis. An additional highly preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease"), and inflammation and inflammatory disorders. Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, melanoma, and/or as described below under "Hyperproliferative</p>
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				<p>contents of each of which are herein incorporated by reference in its entirety. Human T cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human T cells are primary human lymphocytes that mature in the thymus and express a T cell receptor and CD3, CD4, or CD8. These cells mediate humoral or cell-mediated immunity and may be preactivated to enhance responsiveness to immunomodulatory factors.</p>	<p>Disorders"). Preferred indications include neoplasms and cancers, such as, leukemia, lymphoma, melanoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, leukemias, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis,</p>
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24	HCE3G69	328	Inhibition of squalene synthetase gene transcription.	Reporter Assay: construct contains regulatory and coding sequence of squalene synthetase, the first specific enzyme in the cholesterol biosynthetic pathway. See Jiang, et al., J. Biol. Chem. 268:12818-12824(1993), the contents of which are herein incorporated by reference in its entirety. Cells were treated with SID supernatants, and SEAP activity was measured after 72 hours. HepG2 is a human hepatocellular carcinoma cell line (ATCC HB-8065). See Knowles et al., Science. 209:497-9 (1980), the contents of which are herein incorporated by reference in its entirety.	meningitis, and Lyme Disease.
24	HCE3G69	328	Proliferation of pre-adipose cells (such as 3T3-L1 cells)	Assays for the regulation (i.e. increases or decreases) of viability and proliferation of cells in vitro are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including	

				antibodies and agonists or antagonists of the invention) to regulate viability and proliferation of pre-adipose cells and cell lines. For example, the CellTiter-Glo [®] Luminescent Cell Viability Assay (Promega Corp., Madison, WI, USA) can be used to measure the number of viable cells in culture based on quantitation of the ATP present which signals the presence of metabolically active cells. 3T3-L1 is a mouse preadipocyte cell line. It is a continuous substrain of 3T3 fibroblast cells developed through clonal isolation. Cells were differentiated to an adipose-like state before being used in the screen. See Green H and Meuth M., Cell 3: 127-133 (1974), which is herein incorporated by reference in its entirety.	
24	HCE3G69	328	Stimulation of insulin secretion from pancreatic beta cells.	Assays for measuring secretion of insulin are well-known in the art and may be used or routinely modified to assess	A highly preferred indication is diabetes mellitus. An additional highly preferred indication is a complication

				<p>the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate insulin secretion. For example, insulin secretion is measured by FMAT using anti-rat insulin antibodies. Insulin secretion from pancreatic beta cells is upregulated by glucose and also by certain proteins/peptides, and dysregulation is a key component in diabetes. Exemplary assays that may be used or routinely modified to test for stimulation of insulin secretion (from pancreatic cells) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Ahren, B., et al., Am J Physiol, 277(4 Pt 2):R959-66 (1999); Li, M., et al., Endocrinology, 138(9):3735-40 (1997); Kim, K.H., et al., FEBS Lett, 377(2):237-9 (1995); and,</p>	<p>associated with diabetes (e.g., diabetic retinopathy, diabetic nephropathy, kidney disease (e.g., renal failure, nephropathy and/or other diseases and disorders as described in the "Renal Disorders" section below), diabetic neuropathy, nerve disease and nerve damage (e.g., due to diabetic neuropathy), blood vessel blockage, heart disease, stroke, impotence (e.g., due to diabetic neuropathy or blood vessel blockage), seizures, mental confusion, drowsiness, nonketotic hyperglycemic-hypermolar coma, cardiovascular disease (e.g., heart disease, atherosclerosis, microvascular disease, hypertension, stroke, and other diseases and disorders as described in the "Cardiovascular Disorders" section below), dyslipidemia, endocrine disorders (as described in the "Endocrine Disorders" section below),</p>
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				<p>Miraglia S et. al., Journal of Biomolecular Screening, 4:193-204 (1999), the contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include rat INS-1 cells. INS-1 cells are a semi-adherent cell line established from cells isolated from an X-ray induced rat transplantable insulinoma. These cells retain characteristics typical of native pancreatic beta cells including glucose inducible insulin secretion. References: Asfari et al. Endocrinology 1992 130:167.</p>	<p>neuropathy, vision impairment (e.g., diabetic retinopathy and blindness), ulcers and impaired wound healing, and infection (e.g., infectious diseases and disorders as described in the "Infectious Diseases" section below, especially of the urinary tract and skin), carpal tunnel syndrome and Dupuytren's contracture). An additional highly preferred indication is obesity and/or complications associated with obesity. Additional highly preferred indications include weight loss or alternatively, weight gain. Additional highly preferred indications are complications associated with insulin resistance.</p>
24	HCE3G69	328	<p>Production of IL-10 and activation of T-cells.</p>	<p>Assays for production of IL-10 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of</p>	<p>Highly preferred indications include allergy and asthma. Additional highly preferred indications include immune and hematopoietic disorders</p>

				<p>polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays</p>	<p>(e.g., as described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response.</p>
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				include Th2 cells. IL10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.	
25	HCEEA88	329	Inhibition of squalene synthetase gene transcription.	Reporter Assay: construct contains regulatory and coding sequence of squalene synthetase, the first specific enzyme in the cholesterol biosynthetic pathway. See Jiang, et al., J. Biol. Chem. 268:12818-12824(1993), the contents of which are herein incorporated by reference in its entirety. Cells were treated with SID supernatants, and SEAP activity was measured	

25	HCEEA88	329	<p>Production of IL-10 and activation of T-cells.</p>	<p>after 72 hours. HepG2 is a human hepatocellular carcinoma cell line (ATCC HB-8065). See Knowles et al., Science. 209:497-9 (1980), the contents of which are herein incorporated by reference in its entirety.</p> <p>Assays for production of IL-10 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in:</p>	<p>Highly preferred indications include allergy and asthma. Additional highly preferred indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response.</p>
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				<p>Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.</p>
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26	HCEFB69	330	<p>Activation of transcription through GAS response element in immune cells (such as eosinophils).</p>	<p>Assays for the activation of transcription through the Gamma Interferon Activation Site (GAS) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate gene expression (commonly via STAT transcription factors) involved in a wide variety of cell functions. Exemplary assays for transcription through the GAS response element that may be used or routinely modified to test GAS-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988);</p>	<p>Highly preferred indications include asthma, allergy, hypersensitivity reactions, inflammation, and inflammatory disorders. Additional highly preferred indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting an eosinophil-mediated immune response and, alternatively, suppressing an eosinophil-mediated immune response.</p>
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					<p>Matikainen et al., Blood 93(6):1980-1991 (1999); and Henttinen et al., J Immunol 155(10):4582-4587 (1995); the contents of each of which are herein incorporated by reference in its entirety. Moreover, exemplary assays that may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to activate or inhibit activation of immune cells include assays disclosed and/or cited in: Mayumi M., "EoL-1, a human eosinophilic cell line" Leuk Lymphoma; Jun;7(3):243-50 (1992); Bhattacharya S, "Granulocyte macrophage colony-stimulating factor and interleukin-5 activate STAT5 and induce CIS1 mRNA in human peripheral blood eosinophils" Am J Respir Cell Mol Biol; Mar;24(3):312-6 (2001); and, Du J, et al., "Engagement of the CrkL</p>
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				<p>adapter in interleukin-5 signaling in eosinophils" J Biol Chem; Oct 20;275(42):33167-75 (2000); the contents of each of which are herein incorporated by reference in its entirety. Exemplary cells that may be used according to these assays include eosinophils. Eosinophils are a type of immune cell important in the late stage of allergic reactions; they are recruited to tissues and mediate the inflammatory response of late stage allergic reaction. Increases in GAS mediated transcription in eosinophils is typically a result of STAT activation, normally a direct consequence of interleukin or other cytokine receptor stimulation (e.g. IL3, IL5 or GMCSF).</p>	
26	HCEFB69	330	Production of IL-10 and activation of T-cells.	<p>Assays for production of IL-10 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and</p> <p>Highly preferred indications include allergy and asthma. Additional highly preferred indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and</p>	

				agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL10 secreted from Th2 cells may be	"Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response.
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				measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.	
27	HCFMV71	331	Activation of transcription through AP1 response element in immune cells (such as T-cells).	Assays for the activation of transcription through the AP1 response element are known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate growth and other cell functions. Exemplary assays for transcription through the AP1 response element that may be used or routinely modified to test AP1-response	Preferred indications include neoplastic diseases (e.g., as described below under "Hyperproliferative Disorders"), blood disorders (e.g., as described below under "Immune Activity", "Cardiovascular Disorders", and/or "Blood-Related Disorders"), and infection (e.g., an infectious disease as described below under "Infectious Disease"). Highly preferred indications include autoimmune diseases (e.g.,

				<p>element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1988); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Rellahan et al., J Biol Chem 272(49):30806-30811 (1997); Chang et al., Mol Cell Biol 18(9):4986-4993 (1998); and Fraser et al., Eur J Immunol 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety.</p> <p>Mouse T cells that may be used according to these assays are publicly available (e.g., through the ATCC).</p> <p>Exemplary mouse T cells that may be used according to these assays include the HT2 cell line, which is an IL-2 dependent suspension culture cell line that also responds to</p>	<p>rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Additional highly preferred indications include inflammation and inflammatory disorders. Highly preferred indications also include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under “Hyperproliferative Disorders”). Highly preferred indications include neoplasms and cancers, such as, leukemia, lymphoma, prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver, and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include arthritis, asthma, AIDS,</p>
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27	HCFMV71	331	<p>Activation of transcription through cAMP response element in immune cells (such as T-cells).</p>	<p>IL-4.</p>	<p>allergy, anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, granulomatous disease, inflammatory bowel disease, sepsis, psoriasis, suppression of immune reactions to transplanted organs and tissues, endocarditis, meningitis, and Lyme Disease.</p>	<p>Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"), and infection (e.g., an infectious disease as described below under "Infectious Disease"). Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below),</p>
			<p>Assays for the activation of transcription through the cAMP response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to increase cAMP, bind to CREB transcription factor, and modulate expression of genes involved in a wide variety of cell functions. Exemplary assays for transcription through the cAMP response element that</p>			

			<p>may be used or routinely modified to test cAMP-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Black et al., Virus Genes 15(2):105-117 (1997); and Belkowski et al., J Immunol 161(2):659-665 (1998), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the JURKAT cell line, which is a suspension culture of leukemia cells that produce IL-2 when stimulated.</p>	<p>boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional preferred indications include inflammation and inflammatory disorders. Highly preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, leukemia, lymphoma (e.g., T cell lymphoma, Burkitt's lymphoma, non-Hodgkins lymphoma, Hodgkin's disease), melanoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include</p>
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					<p>anemia, pancytopenia, leukopenia, thrombocytopenia, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, and asthma and allergy.</p>
27	HCFMV71	331	<p>Activation of transcription through GAS response element in immune cells (such as T-cells).</p>	<p>Assays for the activation of transcription through the Gamma Interferon Activation Site (GAS) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate STAT transcription factors and modulate gene expression involved in a wide variety of</p>	<p>Highly preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, for example, leukemia, lymphoma (e.g., T cell lymphoma, Burkitt's lymphoma, non-Hodgkins lymphoma, Hodgkin's disease), melanoma, and prostate,</p>

			<p>cell functions. Exemplary assays for transcription through the GAS response element that may be used or routinely modified to test GAS-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Matikainen et al., Blood 93(6):1980-1991 (1999); and Henttinen et al., J Immunol 155(10):4582-4587 (1995), the contents of each of which are herein incorporated by reference in its entirety. Exemplary human T cells, such as the MOL T4 cell line, that may be used according to these assays are publicly available (e.g., through the ATCC).</p>	<p>breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional preferred indications include inflammation and inflammatory disorders. Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"), and infection (e.g., viral</p>
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					infections, tuberculosis, infections associated with chronic granulomatous disease and malignant osteoporosis, and/or an infectious disease as described below under "Infectious Disease"). An additional preferred indication is idiopathic pulmonary fibrosis. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, and asthma and allergy.
27	HCFMV71	331	Activation of transcription through NFKB	Assays for the activation of transcription through the NFKB response element are	Highly preferred indications include inflammation and inflammatory disorders.

			<p>response element in immune cells (such as T-cells).</p>	<p>well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFκB transcription factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription through the NFκB response element that may be used or routinely modified to test NFκB-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Black et al., Virus Gnes 15(2):105-117 (1997); and Fraser et al., 29(3):838-844 (1999), the contents of each of which are</p>	<p>Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), and immunodeficiencies (e.g., as described below). An additional highly preferred indication is infection (e.g., AIDS, and/or an infectious disease as described below under "Infectious Disease"). Highly preferred indications include neoplastic diseases (e.g., melanoma, leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, for example, melanoma, renal cell carcinoma, leukemia,</p>
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				<p>herein incorporated by reference in its entirety. Exemplary human T cells, such as the MOLT4, that may be used according to these assays are publicly available (e.g., through the ATCC).</p>	<p>lymphoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications also include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, suppression of immune reactions to transplanted organs, asthma and allergy.</p>
28	HCNSD93	332	Regulation of apoptosis of immune cells (such	Caspase Apoptosis. Assays for caspase apoptosis are well known in the art and may be	Preferred embodiments of the invention include using polypeptides of the invention

			as mast cells).	<p>used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate caspase protease-mediated apoptosis in immune cells (such as, for example, in mast cells). Mast cells are found in connective and mucosal tissues throughout the body, and their activation via immunoglobulin E - antigen, promoted by T helper cell type 2 cytokines, is an important component of allergic disease. Dysregulation of mast cell apoptosis may play a role in allergic disease and mast cell tumor survival. Exemplary assays for caspase apoptosis that may be used or routinely modified to test caspase apoptosis activity induced by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in: Masuda A, et al., J Biol Chem,</p>	<p>(or antibodies, agonists, or antagonists thereof) in detection, diagnosis, prevention, and/or treatment of asthma, allergy, hypersensitivity and inflammation.</p>
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29	HCUIM65	333	Regulation of transcription via DMEF1 response element in adipocytes and pre-adipocytes	<p>Assays for the regulation of transcription through the DMEF1 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the</p> <p>A highly preferred indication is diabetes mellitus. Additional highly preferred indications include complications associated with diabetes (e.g., diabetic retinopathy, diabetic nephropathy, kidney disease (e.g., renal failure,</p>	

			<p>invention) to activate the DMEF1 response element in a reporter construct (such as that containing the GLUT4 promoter) and to regulate insulin production. The DMEF1 response element is present in the GLUT4 promoter and binds to MEF2 transcription factor and another transcription factor that is required for insulin regulation of Glut4 expression in skeletal muscle. GLUT4 is the primary insulin-responsive glucose transporter in fat and muscle tissue. Exemplary assays that may be used or routinely modified to test for DMEF1 response element activity (in adipocytes and pre-adipocytes) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Thai, M. V., et al., J Biol Chem, 273(23):14285-92 (1998); Mora, S., et al., J Biol Chem, 275(21):16323-8 (2000); Liu, M.L., et al., J Biol</p>	<p>nephropathy and/or other diseases and disorders as described in the "Renal Disorders" section below), diabetic neuropathy, nerve disease and nerve damage (e.g., due to diabetic neuropathy), blood vessel blockage, heart disease, stroke, impotence (e.g., due to diabetic neuropathy or blood vessel blockage), seizures, mental confusion, drowsiness, nonketotic hyperglycemic-hyperosmolar coma, cardiovascular disease (e.g., heart disease, atherosclerosis, microvascular disease, hypertension, stroke, and other diseases and disorders as described in the "Cardiovascular Disorders" section below), dyslipidemia, endocrine disorders (as described in the "Endocrine Disorders" section below), neuropathy, vision impairment (e.g., diabetic retinopathy and blindness), ulcers and impaired wound healing, and infection</p>
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				<p>Chem, 269(45):28514-21 (1994); "Identification of a 30-base pair regulatory element and novel DNA binding protein that regulates the human GLUT4 promoter in transgenic mice", J Biol Chem. 2000 Aug 4;275(31):23666-73; Berger, et al., Gene 66:1-10 (1988); and, Cullen, B., et al., Methods in Enzymol. 216:362-368 (1992), the contents of each of which is herein incorporated by reference in its entirety. Adipocytes and pre-adipocytes that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary cells that may be used according to these assays include the mouse 3T3-L1 cell line which is an adherent mouse preadipocyte cell line. Mouse 3T3-L1 cells are a continuous substrain of 3T3 fibroblasts developed through clonal isolation. These cells</p>	<p>(e.g., infectious diseases and disorders as described in the "Infectious Diseases" section below, especially of the urinary tract and skin). An additional highly preferred indication is obesity and/or complications associated with obesity. Additional highly preferred indications include weight loss or alternatively, weight gain. Additional highly preferred indications are complications associated with insulin resistance.</p>
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29	HCUIM65	333	<p>/</p> <p>Activation of transcription through cAMP response element (CRE) in pre-adipocytes.</p>	<p>undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation culture conditions.</p> <p>Assays for the activation of transcription through the cAMP response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to increase cAMP, regulate CREB transcription factors, and modulate expression of genes involved in a wide variety of cell functions. For example, a 3T3-L1/CRE reporter assay may be used to identify factors that activate the cAMP signaling pathway. CREB plays a major role in adipogenesis, and is involved in differentiation into adipocytes. CRE contains the binding sequence for the transcription factor CREB (CRE binding protein).</p>	<p>A highly preferred indication is obesity and/or complications associated with obesity. Additional highly preferred indications include weight loss or alternatively, weight gain. An additional highly preferred indication is diabetes mellitus. An additional highly preferred indication is a complication associated with diabetes (e.g., diabetic retinopathy, diabetic nephropathy, kidney disease (e.g., renal failure, nephropathy and/or other diseases and disorders as described in the "Renal Disorders" section below), diabetic neuropathy, nerve disease and nerve damage (e.g., due to diabetic neuropathy), blood vessel blockage, heart disease, stroke, impotence (e.g., due to diabetic neuropathy or blood vessel blockage), seizures, mental</p>
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			<p>Exemplary assays for transcription through the cAMP response element that may be used or routinely modified to test cAMP-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Reusch et al., Mol Cell Biol 20(3):1008-1020 (2000); and Klemm et al., J Biol Chem 273:917-923 (1998), the contents of each of which are herein incorporated by reference in its entirety. Pre-adipocytes that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary mouse adipocyte cells that may be used</p>	<p>confusion, drowsiness, nonketotic hyperglycemic-hyperosmolar coma, cardiovascular disease (e.g., heart disease, atherosclerosis, microvascular disease, hypertension, stroke, and other diseases and disorders as described in the "Cardiovascular Disorders" section below), dyslipidemia, endocrine disorders (as described in the "Endocrine Disorders" section below), neuropathy, vision impairment (e.g., diabetic retinopathy and blindness), ulcers and impaired wound healing, and infection (e.g., infectious diseases and disorders as described in the "Infectious Diseases" section below, especially of the urinary tract and skin), carpal tunnel syndrome and Dupuytren's contracture). Additional highly preferred indications are complications associated with insulin resistance.</p>
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29	HCUIM65	333	Activation of transcription through serum response element in pre-adipocytes.	<p>according to these assays include 3T3-L1 cells. 3T3-L1 is an adherent mouse preadipocyte cell line that is a continuous substrain of 3T3 fibroblast cells developed through clonal isolation and undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation conditions known in the art.</p> <p>Assays for the activation of transcription through the Serum Response Element (SRE) are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate the serum response factors and modulate the expression of genes involved in growth. Exemplary assays for transcription through the SRE that may be used or routinely modified to test SRE activity of the polypeptides of the invention (including</p>	<p>A highly preferred indication is obesity and/or complications associated with obesity. Additional highly preferred indications include weight loss or alternatively, weight gain. An additional highly preferred indication is diabetes mellitus. An additional highly preferred indication is a complication associated with diabetes (e.g., diabetic retinopathy, diabetic nephropathy, kidney disease (e.g., renal failure, nephropathy and/or other diseases and disorders as described in the "Renal Disorders" section below), diabetic neuropathy, nerve</p>
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			<p>antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); and Black et al., Virus Genes 12(2):105-117 (1997), the content of each of which are herein incorporated by reference in its entirety. Pre-adipocytes that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary mouse adipocyte cells that may be used according to these assays include 3T3-L1 cells. 3T3-L1 is an adherent mouse preadipocyte cell line that is a continuous substrain of 3T3 fibroblast cells developed through clonal isolation and undergo a pre-adipocyte to adipose-like conversion under</p>	<p>disease and nerve damage (e.g., due to diabetic neuropathy), blood vessel blockage, heart disease, stroke, impotence (e.g., due to diabetic neuropathy or blood vessel blockage), seizures, mental confusion, drowsiness, nonketotic hyperglycemic-hypermolar coma, cardiovascular disease (e.g., heart disease, atherosclerosis, microvascular disease, hypertension, stroke, and other diseases and disorders as described in the "Cardiovascular Disorders" section below), dyslipidemia, endocrine disorders (as described in the "Endocrine Disorders" section below), neuropathy, vision impairment (e.g., diabetic retinopathy and blindness), ulcers and impaired wound healing, and infection (e.g., infectious diseases and disorders as described in the "Infectious Diseases" section below). Additional highly preferred indications are</p>
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				appropriate differentiation conditions known in the art.	complications associated with insulin resistance.
29	HCUIM65	333	Inhibition of squalene synthetase gene transcription.	Reporter Assay: construct contains regulatory and coding sequence of squalene synthetase, the first specific enzyme in the cholesterol biosynthetic pathway. See Jiang, et al., J. Biol. Chem. 268:12818-12824(1993), the contents of which are herein incorporated by reference in its entirety. Cells were treated with SID supernatants, and SEAP activity was measured after 72 hours. HepG2 is a human hepatocellular carcinoma cell line (ATCC HB-8065). See Knowles et al., Science. 209:497-9 (1980), the contents of which are herein incorporated by reference in its entirety.	
29	HCUIM65	333	Stimulation of Calcium Flux in pancreatic beta cells.	Assays for measuring calcium flux are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of	A highly preferred indication is diabetes mellitus. An additional highly preferred indication is a complication associated with diabetes (e.g., diabetic retinopathy, diabetic nephropathy, kidney disease

				<p>the invention) to mobilize calcium. For example, the FLPR assay may be used to measure influx of calcium. Cells normally have very low concentrations of cytosolic calcium compared to much higher extracellular calcium. Extracellular factors can cause an influx of calcium, leading to activation of calcium responsive signaling pathways and alterations in cell functions. Exemplary assays that may be used or routinely modified to measure calcium flux by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Satin LS, et al., Endocrinology, 136(10):4589-601 (1995); Mogami H, et al., Endocrinology, 136(7):2960-6 (1995); Richardson SB, et al., Biochem J, 288 (Pt 3):847-51 (1992); and, Meats, JE, et al., Cell Calcium 1989 Nov-Dec;10(8):535-41 (1989), the contents of each of which is</p> <p>(e.g., renal failure, nephropathy and/or other diseases and disorders as described in the "Renal Disorders" section below), diabetic neuropathy, nerve disease and nerve damage (e.g., due to diabetic neuropathy), blood vessel blockage, heart disease, stroke, impotence (e.g., due to diabetic neuropathy or blood vessel blockage), seizures, mental confusion, drowsiness, nonketotic hyperglycemic-hypermolar coma, cardiovascular disease (e.g., heart disease, atherosclerosis, microvascular disease, hypertension, stroke, and other diseases and disorders as described in the "Cardiovascular Disorders" section below), dyslipidemia, endocrine disorders (as described in the "Endocrine Disorders" section below), neuropathy, vision impairment (e.g., diabetic retinopathy and blindness), ulcers and impaired</p>
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				<p>herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include HIT15 Cells. HIT15 are an adherent epithelial cell line established from Syrian hamster islet cells transformed with SV40. These cells express glucagon, somatostatin, and glucocorticoid receptors. The cells secrete insulin, which is stimulated by glucose and glucagon and suppressed by somatostatin or glucocorticoids. ATTC# CRL-1777 Refs: Lord and Ashcroft. Biochem. J. 219: 547-551; Santerre et al. Proc. Natl. Acad. Sci. USA 78: 4339-4343, 1981.</p>	<p>wound healing, and infection (e.g., infectious diseases and disorders as described in the "Infectious Diseases" section below, especially of the urinary tract and skin), carpal tunnel syndrome and Dupuytren's contracture). An additional highly preferred indication is obesity and/or complications associated with obesity. Additional highly preferred indications include weight loss or alternatively, weight gain. Additional highly preferred indications are complications associated with insulin resistance.</p>
29	HCUIM65	333	Activation of transcription through GATA-3	<p>This reporter assay measures activation of the GATA-3 signaling pathway in HMC-1</p>	<p>Highly preferred indications include allergy, asthma, and rhinitis. Additional preferred</p>

			<p>response element in immune cells (such as mast cells).</p>	<p>human mast cell line. Activation of GATA-3 in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the GATA3 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3 transcription factors and modulate expression of mast cell genes important for immune response development. Exemplary assays for transcription through the GATA3 response element that may be used or routinely modified to test GATA3-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and</p>	<p>indications include infection (e.g., an infectious disease as described below under "Infectious Disease"), and inflammation and inflammatory disorders. Preferred indications also include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, melanoma, prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver, and urinary tract cancers and/or as described below under "Hyperproliferative Disorders"). Other preferred</p>
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HCUIM65	333	Activation of	This reporter assay measures	Highly preferred indications

			transcription through NFAT response element in immune cells (such as mast cells).	activation of the NFAT signaling pathway in HMC-1 human mast cell line. Activation of NFAT in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and	include allergy, asthma, and rhinitis. Additional preferred indications include infection (e.g., an infectious disease as described below under "Infectious Disease"), and inflammation and inflammatory disorders. Preferred indications also include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, melanoma, prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver, and urinary tract cancers and/or as described below under
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				<p>agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Ali et al., J Immunol 165(12):7215-7223 (2000); Hutchinson and McCloskey, J Biol Chem 270(27):16333-16338 (1995), and Turner et al., J Exp Med 188:527-537 (1998), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast</p>	<p>“Hyperproliferative Disorders”). Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, leukemias, Hodgkin’s disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt’s lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, and Lyme Disease.</p>
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29	HCUIM65	333	<p>Activation of transcription through NFKB response element in immune cells (such as mast cells).</p>	<p>cell leukemia, and exhibits many characteristics of immature mast cells.</p> <p>This reporter assay measures activation of the NFKB signaling pathway in HMC-1 human mast cell line.</p> <p>Activation of NFKB in mast cells has been linked to production of certain cytokines, such as IL-6 and IL-9. Assays for the activation of transcription through the NFKB response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription factors and modulate expression of immunomodulatory genes.</p> <p>Exemplary assays for transcription through the NFKB response element that may be used or routinely modified to test NFKB-response element activity of</p>	<p>Highly preferred indication includes allergy, asthma, and rhinitis. Additional highly preferred indications include infection (e.g., an infectious disease as described below under "Infectious Disease"), and inflammation and inflammatory disorders.</p> <p>Preferred indications include immunological and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders").</p> <p>Preferred indications also include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Preferred indications also include neoplastic diseases (e.g., leukemia, lymphoma, melanoma, and/or as described</p>
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				<p>polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Stassen et al, J Immunol 166(7):4391-8 (2001); and Marquardt and Walker, J Allergy Clin Immunol 105(3):500-5 (2000), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of</p>	<p>below under "Hyperproliferative Disorders"). Preferred indications include neoplasms and cancer, such as, for example, leukemia, lymphoma, melanoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver, urinary tract cancers and as described below under "Hyperproliferative Disorders".</p>
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29	HCUIM65	333	<p>Production of VCAM in endothelial cells (such as human umbilical vein endothelial cells (HUVEC))</p>	<p>immature mast cells. Assays for measuring expression of VCAM are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate VCAM expression. For example, FMAAT may be used to measure the upregulation of cell surface VCAM-1 expression in endothelial cells. Endothelial cells are cells that line blood vessels, and are involved in functions that include, but are not limited to, angiogenesis, vascular permeability, vascular tone, and immune cell extravasation. Exemplary endothelial cells that may be used according to these assays include human umbilical vein endothelial cells (HUVEC), which are available from commercial sources. The expression of VCAM (CD106), a membrane-</p>	<p>Highly preferred indications include inflammation (acute and chronic), restnosis, atherosclerosis, asthma and allergy. Highly preferred indications include inflammation and inflammatory disorders, immunological disorders, neoplastic disorders (e.g. cancer/tumorigenesis), and cardiovascular disorders (such as described below under "Immune Activity", "Blood-Related Disorders", "Hyperproliferative Disorders" and/or "Cardiovascular Disorders"). Highly preferred indications include neoplasms and cancers such as, for example, leukemia, lymphoma, melanoma, renal cell carcinoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic</p>
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				associated protein, can be upregulated by cytokines or other factors, and contributes to the extravasation of lymphocytes, leucocytes and other immune cells from blood vessels; thus VCAM expression plays a role in promoting immune and inflammatory responses.	conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia.
29	HCUIM65	333	Activation of transcription through NFAT response element in immune cells (such as natural killer cells).	Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-	Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity," "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional highly preferred indications include inflammation and

				<p>response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Aramburu et al., J Exp Med 182(3):801-810 (1995); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Fraser et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J Biol Chem 268(19):14285-14293 (1993), the contents of each of which are herein incorporated by reference in its entirety. NK cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human NK cells that may be used according to these assays include the NK-YT cell line, which is a human</p>	<p>inflammatory disorders. An additional highly preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease"). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Preferred indications include neoplasms and cancers, such as, for example, leukemia, lymphoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications also include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple</p>
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29	HCUIM65	333	<p>Activation of transcription through NFKB response element in immune cells (such as natural killer cells).</p>	<p>Assays for the activation of transcription through the NFKB response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription through the NFKB response element that may be used or routinely modified to test NFKB-</p>	<p>natural killer cell line with cytolytic and cytotoxic activity.</p>	<p>myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, asthma and allergy.</p>	<p>Highly preferred indications include inflammation and inflammatory disorders. Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), and immunodeficiencies (e.g., as described below). An additional highly preferred</p>
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				<p>response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Valle Blazquez et al, Immunology 90(3):455-460 (1997); Aramburau et al., J Exp Med 82(3):801-810 (1995); and Fraser et al., 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. NK cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary NK cells that may be used according to these assays include the NK-YT cell line, which is a human natural killer cell line with cytolytic and cytotoxic activity.</p>	<p>indication is infection (e.g., AIDS, and/or an infectious disease as described below under "Infectious Disease"). Highly preferred indications include neoplastic diseases (e.g., melanoma, leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, for example, melanoma, renal cell carcinoma, leukemia, lymphoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications also include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL),</p>
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					<p>plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, suppression of immune reactions to transplanted organs, asthma and allergy.</p>
29	HCUIM65	333	<p>Activation of transcription through serum response element in immune cells (such as natural killer cells).</p>	<p>Assays for the activation of transcription through the Serum Response Element (SRE) are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate serum response factors and modulate the expression of genes involved in growth and upregulate the function of growth-related genes in many cell types. Exemplary assays for transcription through the SRE</p>	<p>A preferred embodiment of the invention includes a method for inhibiting (e.g., reducing) TNF alpha production. An alternative highly preferred embodiment of the invention includes a method for stimulating (e.g., increasing) TNF alpha production. Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"), Highly preferred indications include autoimmune diseases</p>

				<p>that may be used or routinely modified to test SRE activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., <i>Gene</i> 66:1-10 (1998); Cullen and Malm, <i>Methods in Enzymol</i> 216:362-368 (1992); Henthorn et al., <i>Proc Natl Acad Sci USA</i> 85:6342-6346 (1988); Benson et al., <i>J Immunol</i> 153(9):3862-3873 (1994); and Black et al., <i>Virus Genes</i> 12(2):105-117 (1997), the content of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary T cells that may be used according to these assays include the NK-YT cell line, which is a human natural killer cell line with cytolytic and cytotoxic activity.</p>	<p>(e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional highly preferred indications include inflammation and inflammatory disorders, and treating joint damage in patients with rheumatoid arthritis. An additional highly preferred indication is sepsis. Highly preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Additionally, highly preferred indications include neoplasms and cancers, such as, for example, leukemia, lymphoma, melanoma, glioma (e.g., malignant glioma), solid tumors, and prostate, breast,</p>
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					lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, cardiac reperfusion injury, and asthma and allergy. An additional preferred indication is infection (e.g., an infectious
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29	HCUIM65	333	<p>Activation of transcription through GAS response element in immune cells (such as T-cells).</p>	<p>Assays for the activation of transcription through the Gamma Interferon Activation Site (GAS) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate STAT transcription factors and modulate gene expression involved in a wide variety of cell functions. Exemplary assays for transcription through the GAS response element that may be used or routinely modified to test GAS-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn</p>	<p>disease as described below under "Infectious Disease").</p> <p>Highly preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, for example, leukemia, lymphoma (e.g., T cell lymphoma, Burkitt's lymphoma, non-Hodgkins lymphoma, Hodgkin's disease), melanoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described</p>
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				<p>et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Matikainen et al., Blood 93(6):1980-1991 (1999); and Henttinen et al., J Immunol 155(10):4582-4587 (1995), the contents of each of which are herein incorporated by reference in its entirety. Exemplary human T cells, such as the SUPT cell line, that may be used according to these assays are publicly available (e.g., through the ATCC).</p>	<p>below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional preferred indications include inflammation and inflammatory disorders. Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"), and infection (e.g., viral infections, tuberculosis, infections associated with chronic granulomatous disease and malignant osteoporosis, and/or an infectious disease as described below under "Infectious Disease"). An additional preferred indication is idiopathic pulmonary fibrosis. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, acute lymphocytic anemia</p>
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30	HCWKC15	334	Regulation of transcription via DMEF1 response element in adipocytes and pre-adipocytes	Assays for the regulation of transcription through the DMEF1 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to activate the DMEF1 response element in a reporter construct (such as that containing the GLUT4 promoter) and to regulate insulin production. The DMEF1 response element is present in the GLUT4	(ALL), plasmacytomas, multiple myeloma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, and asthma and allergy.
				A highly preferred indication is diabetes mellitus. Additional highly preferred indications include complications associated with diabetes (e.g., diabetic retinopathy, diabetic nephropathy, kidney disease (e.g., renal failure, nephropathy and/or other diseases and disorders as described in the "Renal Disorders" section below), diabetic neuropathy, nerve disease and nerve damage (e.g., due to diabetic neuropathy), blood vessel	

				<p>promoter and binds to MEF2 transcription factor and another transcription factor that is required for insulin regulation of Glut4 expression in skeletal muscle. GLUT4 is the primary insulin-responsive glucose transporter in fat and muscle tissue. Exemplary assays that may be used or routinely modified to test for DMEF1 response element activity (in adipocytes and pre-adipocytes) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Thai, M.V., et al., J Biol Chem, 273(23):14285-92 (1998); Mora, S., et al., J Biol Chem, 275(21):16323-8 (2000); Liu, M.L., et al., J Biol Chem, 269(45):28514-21 (1994); "Identification of a 30-base pair regulatory element and novel DNA binding protein that regulates the human GLUT4 promoter in transgenic mice", J Biol Chem. 2000 Aug 4;275(31):23666-73;</p>	<p>blockage, heart disease, stroke, impotence (e.g., due to diabetic neuropathy or blood vessel blockage), seizures, mental confusion, drowsiness, nonketotic hyperglycemic-hyposmolar coma, cardiovascular disease (e.g., heart disease, atherosclerosis, microvascular disease, hypertension, stroke, and other diseases and disorders as described in the "Cardiovascular Disorders" section below), dyslipidemia, endocrine disorders (as described in the "Endocrine Disorders" section below), neuropathy, vision impairment (e.g., diabetic retinopathy and blindness), ulcers and impaired wound healing, and infection (e.g., infectious diseases and disorders as described in the "Infectious Diseases" section below, especially of the urinary tract and skin). An additional highly preferred indication is obesity and/or complications associated with</p>
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				<p>Berger, et al., Gene 66:1-10 (1988); and, Cullen, B., et al., Methods in Enzymol. 216:362-368 (1992), the contents of each of which is herein incorporated by reference in its entirety.</p> <p>Adipocytes and pre-adipocytes that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated.</p> <p>Exemplary cells that may be used according to these assays include the mouse 3T3-L1 cell line which is an adherent mouse preadipocyte cell line.</p> <p>Mouse 3T3-L1 cells are a continuous substrain of 3T3 fibroblasts developed through clonal isolation. These cells undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation culture conditions.</p> <p>Assays for the activation of transcription through the cAMP response element are well-known in the art and may</p>	<p>obesity. Additional highly preferred indications include weight loss or alternatively, weight gain. Additional highly preferred indications are complications associated with insulin resistance.</p>
30	HCWKC15	334	Activation of transcription through cAMP response element	<p>A highly preferred indication is obesity and/or complications associated with obesity. Additional highly preferred</p>	

		(CRE) in pre-adipocytes.	<p>be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to increase cAMP, regulate CREB transcription factors, and modulate expression of genes involved in a wide variety of cell functions. For example, a 3T3-L1/CRE reporter assay may be used to identify factors that activate the cAMP signaling pathway. CREB plays a major role in adipogenesis, and is involved in differentiation into adipocytes. CRE contains the binding sequence for the transcription factor CREB (CRE binding protein). Exemplary assays for transcription through the cAMP response element that may be used or routinely modified to test cAMP-response element activity of polypeptides of the invention (including antibodies and</p>	<p>indications include weight loss or alternatively, weight gain. An additional highly preferred indication is diabetes mellitus. An additional highly preferred indication is a complication associated with diabetes (e.g., diabetic retinopathy, diabetic nephropathy, kidney disease (e.g., renal failure, nephropathy and/or other diseases and disorders as described in the "Renal Disorders" section below), diabetic neuropathy, nerve disease and nerve damage (e.g., due to diabetic neuropathy), blood vessel blockage, heart disease, stroke, impotence (e.g., due to diabetic neuropathy or blood vessel blockage), seizures, mental confusion, drowsiness, nonketotic hyperglycemic-hyperosmolar coma, cardiovascular disease (e.g., heart disease, atherosclerosis, microvascular disease, hypertension, stroke, and other diseases and disorders as</p>
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				<p>agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Reusch et al., Mol Cell Biol 20(3):1008-1020 (2000); and Klemm et al., J Biol Chem 273:917-923 (1998), the contents of each of which are herein incorporated by reference in its entirety. Pre-adipocytes that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary mouse adipocyte cells that may be used according to these assays include 3T3-L1 cells. 3T3-L1 is an adherent mouse preadipocyte cell line that is a continuous substrain of 3T3 fibroblast cells developed through clonal isolation and undergo a pre-adipocyte to</p>	<p>described in the "Cardiovascular Disorders" section below), dyslipidemia, endocrine disorders (as described in the "Endocrine Disorders" section below), neuropathy, vision impairment (e.g., diabetic retinopathy and blindness), ulcers and impaired wound healing, and infection (e.g., infectious diseases and disorders as described in the "Infectious Diseases" section below, especially of the urinary tract and skin), carpal tunnel syndrome and Dupuytren's contracture). Additional highly preferred indications are complications associated with insulin resistance.</p>
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30	HCWKC15	334	Activation of transcription through serum response element in pre-adipocytes.	adipose-like conversion under appropriate differentiation conditions known in the art. Assays for the activation of transcription through the Serum Response Element (SRE) are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate the serum response factors and modulate the expression of genes involved in growth. Exemplary assays for transcription through the SRE that may be used or routinely modified to test SRE activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA	A highly preferred indication is obesity and/or complications associated with obesity. Additional highly preferred indications include weight loss or alternatively, weight gain. An additional highly preferred indication is diabetes mellitus. An additional highly preferred indication is a complication associated with diabetes (e.g., diabetic retinopathy, diabetic nephropathy, kidney disease (e.g., renal failure, nephropathy and/or other diseases and disorders as described in the "Renal Disorders" section below), diabetic neuropathy, nerve disease and nerve damage (e.g., due to diabetic neuropathy), blood vessel blockage, heart disease, stroke, impotence (e.g., due to diabetic neuropathy or blood vessel blockage), seizures, mental confusion, drowsiness,
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			<p>85:6342-6346 (1988); and Black et al., Virus Genes 12(2):105-117 (1997), the content of each of which are herein incorporated by reference in its entirety. Pre-adipocytes that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary mouse adipocyte cells that may be used according to these assays include 3T3-L1 cells. 3T3-L1 is an adherent mouse preadipocyte cell line that is a continuous substrain of 3T3 fibroblast cells developed through clonal isolation and undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation conditions known in the art.</p>	<p>nonketotic hyperglycemic-hyperosmolar coma, cardiovascular disease (e.g., heart disease, atherosclerosis, microvascular disease, hypertension, stroke, and other diseases and disorders as described in the "Cardiovascular Disorders" section below), dyslipidemia, endocrine disorders (as described in the "Endocrine Disorders" section below), neuropathy, vision impairment (e.g., diabetic retinopathy and blindness), ulcers and impaired wound healing, and infection (e.g., infectious diseases and disorders as described in the "Infectious Diseases" section below). Additional highly preferred indications are complications associated with insulin resistance.</p>
30	HCWKC15	334	<p>Assays for the activation of transcription through the Gamma Interferon Activation Site (GAS) response element are well-known in the art and may be used or routinely</p> <p>Activation of transcription through GAS response element in immune cells (such as eosinophils).</p>	<p>Highly preferred indications include asthma, allergy, hypersensitivity reactions, inflammation, and inflammatory disorders. Additional highly preferred</p>

				<p>modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate gene expression (commonly via STAT transcription factors) involved in a wide variety of cell functions. Exemplary assays for transcription through the GAS response element that may be used or routinely modified to test GAS-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Matikainen et al., Blood 93(6):1980-1991 (1999); and Henttinen et al., J Immunol 155(10):4582-4587 (1995); the contents of each of which are herein incorporated by</p>	<p>indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting an eosinophil-mediated immune response and, alternatively, suppressing an eosinophil-mediated immune response.</p>
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				<p>reference in its entirety.</p> <p>Moreover, exemplary assays that may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to activate or inhibit activation of immune cells include assays disclosed and/or cited in: Mayumi M., "EoL-1, a human eosinophilic cell line" Leuk Lymphoma; Jun;7(3):243-50 (1992); Bhattacharya S, "Granulocyte macrophage colony-stimulating factor and interleukin-5 activate STAT5 and induce CIS1 mRNA in human peripheral blood eosinophils" Am J Respir Cell Mol Biol; Mar;24(3):312-6 (2001); and, Du J, et al., "Engagement of the CrkL adapter in interleukin-5 signaling in eosinophils" J Biol Chem; Oct 20;275(42):33167-75 (2000); the contents of each of which are herein incorporated by reference in its</p>	
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				<p>entirety. Exemplary cells that may be used according to these assays include eosinophils. Eosinophils are a type of immune cell important in the late stage of allergic reactions; they are recruited to tissues and mediate the inflammatory response of late stage allergic reaction. Increases in GAS mediated transcription in eosinophils is typically a result of STAT activation, normally a direct consequence of interleukin or other cytokine receptor stimulation (e.g. IL3, IL5 or GMCSF).</p>	
30	HCWKC15	334	<p>Activation of transcription through NFKB response element in immune cells (such as EOL1 cells).</p>	<p>Assays for the activation of transcription through the NFKB response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription factors and modulate expression of immunomodulatory genes.</p>	<p>Highly preferred indications include asthma, allergy, hypersensitivity reactions, and inflammation. Preferred indications include infection (e.g., an infectious disease as described below under "Infectious Disease"), immunological disorders, inflammation and inflammatory disorders (e.g., as described below under "Immune Activity", and</p>

				<p>Exemplary assays for transcription through the NFkB response element that may be used or routinely modified to test NFkB-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., <i>Gene</i> 66:1-10 (1998); Cullen and Malm, <i>Methods in Enzymol</i> 216:362-368 (1992); Henthorn et al., <i>Proc Natl Acad Sci USA</i> 85:6342-6346 (1988); Valle Blazquez et al, <i>Immunology</i> 90(3):455-460 (1997); Aramburau et al., <i>J Exp Med</i> 82(3):801-810 (1995); and Fraser et al., 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. For example, a reporter assay (which measures increases in transcription inducible from a NFkB responsive element in EOL-1 cells) may link the NFkB element to a reporter</p>	<p>“Blood-Related Disorders”). Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below).</p>
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				gene and binds to the NFkB transcription factor, which is upregulated by cytokines and other factors. Exemplary immune cells that may be used according to these assays include eosinophils such as the human EOL-1 cell line of eosinophils. Eosinophils are a type of immune cell important in the allergic responses; they are recruited to tissues and mediate the inflammatory response of late stage allergic reaction. Eol-1 is a human eosinophil cell line.	
30	HCWKC15	334	Activation of transcription through GATA-3 response element in immune cells (such as mast cells).	<p>This reporter assay measures activation of the GATA-3 signaling pathway in HMC-1 human mast cell line.</p> <p>Activation of GATA-3 in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the GATA3 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of</p>	<p>Highly preferred indications include allergy, asthma, and rhinitis. Additional preferred indications include infection (e.g., an infectious disease as described below under "Infectious Disease"), and inflammation and inflammatory disorders.</p> <p>Preferred indications also include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or</p>

				<p>the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3 transcription factors and modulate expression of mast cell genes important for immune response development. Exemplary assays for transcription through the GATA3 response element that may be used or routinely modified to test GATA3-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Flavell et al., Cold Spring Harb Symp Quant Biol 64:563-571 (1999); Rodriguez-Palmero et al., Eur J Immunol 29(12):3914-3924 (1999); Zheng and Flavell, Cell 89(4):587-596 (1997); and Henderson et al., Mol Cell Biol</p>	<p>"Cardiovascular Disorders"). Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, melanoma, prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver, and urinary tract cancers and/or as described below under "Hyperproliferative Disorders"). Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, leukemias, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas,</p>
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				<p>14(6):4286-4294 (1994), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.</p>	<p>multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, and Lyme Disease.</p>
30	HCWKC15	334	<p>Activation of transcription through NFAT response element in immune cells (such as mast cells).</p>	<p>This reporter assay measures activation of the NFAT signaling pathway in HMC-1 human mast cell line. Activation of NFAT in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-</p>	<p>Highly preferred indications include allergy, asthma, and rhinitis. Additional preferred indications include infection (e.g., an infectious disease as described below under "Infectious Disease"), and inflammation and inflammatory disorders. Preferred indications also include blood disorders (e.g., as described below under</p>

				<p>known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Ali et al., J Immunol</p>	<p>“Immune Activity”, “Blood-Related Disorders”, and/or “Cardiovascular Disorders”). Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, melanoma, prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver, and urinary tract cancers and/or as described below under “Hyperproliferative Disorders”). Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, leukemias, Hodgkin’s disease,</p>
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				<p>165(12):7215-7223 (2000); Hutchinson and McCloskey, J Biol Chem 270(27):16333-16338 (1995), and Turner et al., J Exp Med 188:527-537 (1998), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.</p>	<p>acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, and Lyme Disease.</p>
30	HCWKC15	334	<p>Activation of transcription through NFkB response element in immune cells (such as mast cells).</p>	<p>This reporter assay measures activation of the NFkB signaling pathway in HMC-1 human mast cell line. Activation of NFkB in mast cells has been linked to production of certain cytokines, such as IL-6 and IL-</p>	<p>Highly preferred indication includes allergy, asthma, and rhinitis. Additional highly preferred indications include infection (e.g., an infectious disease as described below under "Infectious Disease"), and inflammation and</p>

				<p>9. Assays for the activation of transcription through the NFKB response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription through the NFKB response element that may be used or routinely modified to test NFKB-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Stassen et al, J Immunol 166(7):4391-8</p>	<p>inflammatory disorders. Preferred indications include immunological and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"). Preferred indications also include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Preferred indications also include neoplastic diseases (e.g., leukemia, lymphoma, melanoma, and/or as described below under "Hyperproliferative Disorders"). Preferred indications include neoplasms and cancer, such as, for example, leukemia, lymphoma, melanoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver, urinary tract cancers and as described below under</p>
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				<p>(2001); and Marquardt and Walker, J Allergy Clin Immunol 105(3):500-5 (2000), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.</p>	<p>"Hyperproliferative Disorders".</p>
30	HCWKC15	334	<p>Activation of transcription through STAT6 response element in immune cells (such as mast cells).</p>	<p>Assays for the activation of transcription through the Signal Transducers and Activators of Transcription (STAT6) response element in immune cells (such as in the human HMC-1 mast cell line) are well-known in the art and may be used or routinely modified to assess the ability</p>	<p>Highly preferred indications include allergy, asthma, and rhinitis. Additional highly preferred indications include infection (e.g., an infectious disease as described below under "Infectious Disease"), and inflammation and inflammatory disorders. Preferred indications also</p>

			<p>of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate STAT6 transcription factors and modulate the expression of multiple genes. Exemplary assays for transcription through the STAT6 response element that may be used or routinely modified to test STAT6 response element activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Sherman, Immunol Rev 179:48-56 (2001); Malaviya and Uckun, J Immunol 168:421-426 (2002); Masuda et al., J Biol Chem 275(38):29331-29337 (2000); and Masuda et al., J Biol Chem</p>	<p>include hematopoietic and immunological disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), and immunodeficiencies (e.g., as described below). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, melanoma, and/or as described below under "Hyperproliferative Disorders"). Preferred indications include neoplasms and cancer, such as, for example, leukemia, lymphoma, melanoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for</p>
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				<p>276:26107-26113 (2001), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.</p>	<p>example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include hematopoietic and immunological disorders such as arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, and Lyme Disease.</p>
30	HCWKC15	334	<p>Activation of transcription through NFkB response element in immune cells (such as basophils).</p>	<p>This reporter assay measures activation of the NFkB signaling pathway in Ku812 human basophil cell line. Assays for the activation of transcription through the NFkB response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and</p>	<p>Highly preferred indication includes allergy, asthma, and rhinitis. Additional highly preferred indications include infection (e.g., an infectious disease as described below under "Infectious Disease"), and inflammation and inflammatory disorders. Preferred indications include immunological and hematopoietic disorders (e.g.,</p>

				<p>agonists or antagonists of the invention) to regulate NFkB transcription factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription through the NFkB response element that may be used or routinely modified to test NFkB-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Marone et al, Int Arch Allergy Immunol 114(3):207-17 (1997), the contents of each of which are herein incorporated by reference in its entirety. Basophils that may be used according to these assays are publicly available (e.g., through the ATCC).</p>	<p>as described below under "Immune Activity", and "Blood-Related Disorders"). Preferred indications also include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Preferred indications also include neoplastic diseases (e.g., leukemia, lymphoma, melanoma, and/or as described below under "Hyperproliferative Disorders"). Preferred indications include neoplasms and cancer, such as, for example, leukemia, lymphoma, melanoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver, urinary tract cancers and as described below under "Hyperproliferative Disorders".</p>
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30	HCWKC15	334	<p>Activation of transcription through NFAT response element in immune cells (such as natural killer cells).</p>	<p>Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-</p>	<p>Exemplary human basophil cell lines that may be used according to these assays include Ku812, originally established from a patient with chronic myelogenous leukemia. It is an immature prebasophilic cell line that can be induced to differentiate into mature basophils.</p>	<p>Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional highly preferred indications include inflammation and</p>
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			<p>response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Aramburu et al., J Exp Med 182(3):801-810 (1995); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Fraser et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J Biol Chem 268(19):14285-14293 (1993), the contents of each of which are herein incorporated by reference in its entirety. NK cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human NK cells that may be used according to these assays include the NK-YT cell line, which is a human</p>	<p>inflammatory disorders. An additional highly preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease"). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Preferred indications include neoplasms and cancers, such as, for example, leukemia, lymphoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications also include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple</p>
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				natural killer cell line with cytolytic and cytotoxic activity.	myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, asthma and allergy.
30	HCWKC15	334	Activation of transcription through NFKB response element in immune cells (such as natural killer cells).	Assays for the activation of transcription through the NFKB response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription through the NFKB response element that may be used or routinely modified to test NFKB-	Highly preferred indications include inflammation and inflammatory disorders. Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), and immunodeficiencies (e.g., as described below). An additional highly preferred

				<p>response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Valle Blazquez et al, Immunology 90(3):455-460 (1997); Aramburau et al., J Exp Med 82(3):801-810 (1995); and Fraser et al., 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. NK cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary NK cells that may be used according to these assays include the NK-YT cell line, which is a human natural killer cell line with cytolytic and cytotoxic activity.</p>	<p>indication is infection (e.g., AIDS, and/or an infectious disease as described below under "Infectious Disease"). Highly preferred indications include neoplastic diseases (e.g., melanoma, leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, for example, melanoma, renal cell carcinoma, leukemia, lymphoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications also include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL),</p>
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					<p>plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, suppression of immune reactions to transplanted organs, asthma and allergy.</p>
30	HCWKC15	334	<p>Activation of transcription through serum response element in immune cells (such as natural killer cells).</p>	<p>Assays for the activation of transcription through the Serum Response Element (SRE) are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate serum response factors and modulate the expression of genes involved in growth and upregulate the function of growth-related genes in many cell types. Exemplary assays for transcription through the SRE</p>	<p>A preferred embodiment of the invention includes a method for inhibiting (e.g., reducing) TNF alpha production. An alternative highly preferred embodiment of the invention includes a method for stimulating (e.g., increasing) TNF alpha production. Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Highly preferred indications include autoimmune diseases</p>

				<p>that may be used or routinely modified to test SRE activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Benson et al., J Immunol 153(9):3862-3873 (1994); and Black et al., Virus Genes 12(2):105-117 (1997), the content of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary T cells that may be used according to these assays include the NK-YT cell line, which is a human natural killer cell line with cytolytic and cytotoxic activity.</p>	<p>(e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional highly preferred indications include inflammation and inflammatory disorders, and treating joint damage in patients with rheumatoid arthritis. An additional highly preferred indication is sepsis. Highly preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Additionally, highly preferred indications include neoplasms and cancers, such as, for example, leukemia, lymphoma, melanoma, glioma (e.g., malignant glioma), solid tumors, and prostate, breast,</p>
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					lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, cardiac reperfusion injury, and asthma and allergy. An additional preferred indication is infection (e.g., an infectious
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30	HCWKC15	334	<p>Activation of transcription through NFKB response element in immune cells (such as natural killer cells).</p>	<p>Assays for the activation of transcription through the NFKB response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription through the NFKB response element that may be used or routinely modified to test NFKB-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA</p>	<p>disease as described below under "Infectious Disease").</p> <p>Highly preferred indications include inflammation and inflammatory disorders. Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), and immunodeficiencies (e.g., as described below). An additional highly preferred indication is infection (e.g., AIDS, and/or an infectious disease as described below under "Infectious Disease"). Highly preferred indications include neoplastic diseases (e.g., melanoma, leukemia, lymphoma, and/or as described below under "Hyperproliferative</p>
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			<p>85:6342-6346 (1988); Valle Blazquez et al, Immunology 90(3):455-460 (1997); Aramburau et al., J Exp Med 82(3):801-810 (1995); and Fraser et al., 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. NK cells that may be used according to these assays are publicly available (e.g., through the ATCC).</p> <p>Exemplary human NK cells that may be used according to these assays include the NK cell line, which is a human natural killer cell line established from the peripheral blood of a patient with large granular lymphocytic leukemia. This IL-2 dependent suspension culture cell line has a morphology resembling that of activated NK cells.</p>	<p>Disorders”). Highly preferred indications include neoplasms and cancers, such as, for example, melanoma, renal cell carcinoma, leukemia, lymphoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications also include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, suppression of immune</p>
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					reactions to transplanted organs, asthma and allergy.
30	HCWKC15	334	Activation of transcription through AP1 response element in immune cells (such as T-cells).	<p>Assays for the activation of transcription through the AP1 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate growth and other cell functions. Exemplary assays for transcription through the AP1 response element that may be used or routinely modified to test AP1-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1988); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Rellahan et al., J Biol Chem 272(49):30806-30811 (1997);</p>	<p>Preferred indications include neoplastic diseases (e.g., as described below under "Hyperproliferative Disorders"), blood disorders (e.g., as described below under "Immune Activity", "Cardiovascular Disorders", and/or "Blood-Related Disorders"), and infection (e.g., an infectious disease as described below under "Infectious Disease"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Additional highly preferred indications include inflammation and inflammatory disorders. Highly preferred indications also include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described</p>

				<p>Chang et al., Mol Cell Biol 18(9):4986-4993 (1998); and Fraser et al., Eur J Immunol 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. Human T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is an IL-2 and IL-4 responsive suspension-culture cell line.</p>	<p>below under “Hyperproliferative Disorders”). Highly preferred indications include neoplasms and cancers, such as, leukemia, lymphoma, prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver, and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include arthritis, asthma, AIDS, allergy, anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin’s disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt’s lymphoma, granulomatous disease, inflammatory bowel disease, sepsis, psoriasis, suppression of immune reactions to transplanted organs and tissues, endocarditis, meningitis, and Lyme Disease.</p>
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30	HCWKC15	334	<p>Activation of transcription through CD28 response element in immune cells (such as T-cells).</p>	<p>Assays for the activation of transcription through the CD28 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate IL-2 expression in T cells. Exemplary assays for transcription through the CD28 response element that may be used or routinely modified to test CD28-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); McGuire and Iacobelli, J Immunol 159(3):1319-1327 (1997); Parra et al., J Immunol 166(4):2437-2443 (2001); and</p>	<p>A highly preferred embodiment of the invention includes a method for stimulating T cell proliferation. An alternative highly preferred embodiment of the invention includes a method for inhibiting T cell proliferation. A highly preferred embodiment of the invention includes a method for activating T cells. An alternative highly preferred embodiment of the invention includes a method for inhibiting the activation of and/or inactivating T cells. A highly preferred embodiment of the invention includes a method for stimulating (e.g., increasing) IL-2 production. An alternative highly preferred embodiment of the invention includes a method for inhibiting (e.g., reducing) IL-2 production. Additional highly preferred indications include inflammation and inflammatory disorders.</p>
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			<p>Butscher et al., J Biol Chem 3(1):552-560 (1998), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.</p>	<p>Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Highly preferred indications include neoplastic diseases (e.g., melanoma, renal cell carcinoma, leukemia, lymphoma, and/or as described below under “Hyperproliferative Disorders”). Highly preferred indications include neoplasms and cancers, such as, for example, melanoma (e.g., metastatic melanoma), renal cell carcinoma (e.g., metastatic renal cell carcinoma), leukemia, lymphoma (e.g., T cell lymphoma), and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other</p>
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					<p>preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. A highly preferred indication includes infection (e.g., AIDS, tuberculosis, infections associated with granulomatous disease, and osteoporosis, and/or as described below under "Infectious Disease"). A highly preferred indication is AIDS. Additional highly preferred indications include suppression of immune reactions to transplanted organs and/or tissues, uveitis, psoriasis, and tropical spastic paraparesis. Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Preferred indications also include anemia, pancytopenia, leukopenia, thrombocytopenia,</p>
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					<p>Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, asthma and allergy.</p>
30	HCWKC15	334	<p>Activation of transcription through GAS response element in immune cells (such as T-cells).</p>	<p>Assays for the activation of transcription through the Gamma Interferon Activation Site (GAS) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate STAT transcription factors and modulate gene expression involved in a wide variety of cell functions. Exemplary assays for transcription through the GAS response element that may be used or</p>	<p>Highly preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, for example, leukemia, lymphoma (e.g., T cell lymphoma, Burkitt's lymphoma, non-Hodgkins lymphoma, Hodgkin's disease), melanoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include</p>

				<p>routinely modified to test GAS-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Matikainen et al., Blood 93(6):1980-1991 (1999); and Henttinen et al., J Immunol 155(10):4582-4587 (1995), the contents of each of which are herein incorporated by reference in its entirety. Exemplary human T cells, such as the SUPT cell line, that may be used according to these assays are publicly available (e.g., through the ATCC).</p>	<p>benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional preferred indications include inflammation and inflammatory disorders. Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"), and infection (e.g., viral infections, tuberculosis, infections associated with chronic granulomatous disease and malignant</p>
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			<p>of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Serfling et al., Biochim Biophys Acta 1498(1):1-18 (2000); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Fraser et al., Eur J Immunol 29(3):838-844 (1999); and</p>	<p>include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional highly preferred indications include inflammation and inflammatory disorders. An additional highly preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease"). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Preferred indications include neoplasms and cancers, such as, for example, leukemia, lymphoma, and prostate, breast, lung, colon, pancreatic, esophageal,</p>
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				<p>Yeseen et al., J Biol Chem 268(19):14285-14293 (1993), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.</p>	<p>stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications also include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, asthma and allergy.</p>
30	HCWKC15	334	<p>Activation of transcription through NFKB response element in immune cells (such</p>	<p>Assays for the activation of transcription through the NFKB response element are well-known in the art and may be used or routinely modified</p>	<p>Highly preferred indications include inflammation and inflammatory disorders. Highly preferred indications include blood disorders (e.g.,</p>

			as T-cells).	<p>to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription through the NFKB response element that may be used or routinely modified to test NFKB-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Black et al., Virus Gnes 15(2):105-117 (1997); and Fraser et al., 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. T</p>	<p>as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), and immunodeficiencies (e.g., as described below). An additional highly preferred indication is infection (e.g., AIDS, and/or an infectious disease as described below under "Infectious Disease"). Highly preferred indications include neoplastic diseases (e.g., melanoma, leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, melanoma, renal cell carcinoma, leukemia, lymphoma, and prostate, breast, lung, colon, pancreatic,</p>
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				<p>cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.</p>	<p>esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications also include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, suppression of immune reactions to transplanted organs, asthma and allergy.</p>
31	HCWLD74	335	<p>Activation of transcription through cAMP response element</p>	<p>Assays for the activation of transcription through the cAMP response element are well-known in the art and may</p>	<p>A highly preferred indication is obesity and/or complications associated with obesity. Additional highly preferred</p>

		(CRE) in pre-adipocytes.	<p>be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to increase cAMP, regulate CREB transcription factors, and modulate expression of genes involved in a wide variety of cell functions. For example, a 3T3-L1/CRE reporter assay may be used to identify factors that activate the cAMP signaling pathway. CREB plays a major role in adipogenesis, and is involved in differentiation into adipocytes. CRE contains the binding sequence for the transcription factor CREB (CRE binding protein). Exemplary assays for transcription through the cAMP response element that may be used or routinely modified to test cAMP-response element activity of polypeptides of the invention (including antibodies and</p>	<p>indications include weight loss or alternatively, weight gain. An additional highly preferred indication is diabetes mellitus. An additional highly preferred indication is a complication associated with diabetes (e.g., diabetic retinopathy, diabetic nephropathy, kidney disease (e.g., renal failure, nephropathy and/or other diseases and disorders as described in the "Renal Disorders" section below), diabetic neuropathy, nerve disease and nerve damage (e.g., due to diabetic neuropathy), blood vessel blockage, heart disease, stroke, impotence (e.g., due to diabetic neuropathy or blood vessel blockage), seizures, mental confusion, drowsiness, nonketotic hyperglycemic-hyperosmolar coma, cardiovascular disease (e.g., heart disease, atherosclerosis, microvascular disease, hypertension, stroke, and other diseases and disorders as</p>
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				<p>agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Reusch et al., Mol Cell Biol 20(3):1008-1020 (2000); and Klemm et al., J Biol Chem 273:917-923 (1998), the contents of each of which are herein incorporated by reference in its entirety. Pre-adipocytes that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary mouse adipocyte cells that may be used according to these assays include 3T3-L1 cells. 3T3-L1 is an adherent mouse preadipocyte cell line that is a continuous substrain of 3T3 fibroblast cells developed through clonal isolation and undergo a pre-adipocyte to</p>	<p>described in the "Cardiovascular Disorders" section below), dyslipidemia, endocrine disorders (as described in the "Endocrine Disorders" section below), neuropathy, vision impairment (e.g., diabetic retinopathy and blindness), ulcers and impaired wound healing, and infection (e.g., infectious diseases and disorders as described in the "Infectious Diseases" section below, especially of the urinary tract and skin), carpal tunnel syndrome and Dupuytren's contracture). Additional highly preferred indications are complications associated with insulin resistance.</p>
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31	HCWLD74	335	<p>Activation of transcription through GATA-3 response element in immune cells (such as mast cells).</p>	<p>adipose-like conversion under appropriate differentiation conditions known in the art.</p> <p>This reporter assay measures activation of the GATA-3 signaling pathway in HMC-1 human mast cell line.</p> <p>Activation of GATA-3 in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the GATA3 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3 transcription factors and modulate expression of mast cell genes important for immune response development. Exemplary assays for transcription through the GATA3 response element that may be used or routinely modified to test GATA3-response element</p>	<p>Highly preferred indications include allergy, asthma, and rhinitis. Additional preferred indications include infection (e.g., an infectious disease as described below under "Infectious Disease"), and inflammation and inflammatory disorders.</p> <p>Preferred indications also include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders").</p> <p>Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, melanoma, prostate, breast, lung, colon,</p>
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				<p>activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Flavell et al., Cold Spring Harb Symp Quant Biol 64:563-571 (1999); Rodriguez-Palmero et al., Eur J Immunol 29(12):3914-3924 (1999); Zheng and Flavell, Cell 89(4):587-596 (1997); and Henderson et al., Mol Cell Biol 14(6):4286-4294 (1994), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line</p>	<p>pancreatic, esophageal, stomach, brain, liver, and urinary tract cancers and/or as described below under "Hyperproliferative Disorders"). Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, leukemias, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, and Lyme Disease.</p>
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31	HCWLD74	335	<p>Activation of transcription through NFAT response element in immune cells (such as mast cells).</p>	<p>established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.</p> <p>This reporter assay measures activation of the NFAT signaling pathway in HMC-1 human mast cell line.</p> <p>Activation of NFAT in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the</p>	<p>Highly preferred indications include allergy, asthma, and rhinitis. Additional preferred indications include infection (e.g., an infectious disease as described below under "Infectious Disease"), and inflammation and inflammatory disorders.</p> <p>Preferred indications also include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders").</p> <p>Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Preferred indications include neoplastic diseases (e.g., leukemia,</p>
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				<p>NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Ali et al., J Immunol 165(12):7215-7223 (2000); Hutchinson and McCloskey, J Biol Chem 270(27):16333-16338 (1995), and Turner et al., J Exp Med 188:527-537 (1998), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells</p>	<p>lymphoma, melanoma, prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver, and urinary tract cancers and/or as described below under "Hyperproliferative Disorders"). Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, leukemias, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis,</p>
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				that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.	meningitis, and Lyme Disease.
31	HCWLD74	335	Activation of transcription through cAMP response element in immune cells (such as T-cells).	Assays for the activation of transcription through the cAMP response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to increase cAMP, regulate CREB transcription factors, and modulate expression of genes involved in a wide variety of cell functions. Exemplary assays for transcription through the cAMP response element that may be used or routinely modified to test cAMP-response element activity of polypeptides of the invention	Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"), and infection (e.g., an infectious disease as described below under "Infectious Disease"). Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional

				<p>(including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Black et al., Virus Genes 15(2):105-117 (1997); and Belkowski et al., J Immunol 161(2):659-665 (1998), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary mouse T cells that may be used according to these assays include the HT2 cell line, which is a suspension culture of IL-2 dependent T cells that also respond to IL-4.</p>	<p>preferred indications include inflammation and inflammatory disorders. Highly preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, for example, leukemia, lymphoma (e.g., T cell lymphoma, Burkitt's lymphoma, non-Hodgkins lymphoma, Hodgkin's disease), melanoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, acute lymphocytic anemia</p>
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					(ALL), plasmacytomas, multiple myeloma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, and asthma and allergy.
31	HCWLD74	335	TNF α in Human T-cell 293T	Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions.	Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune
31	HCWLD74	335	Activation of transcription through NFAT response element in immune cells (such as natural killer cells).		

			<p>Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Aramburu et al., J Exp Med 182(3):801-810 (1995); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Fraser et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J Biol Chem 268(19):14285-14293 (1993), the contents of each of which are herein incorporated by reference in its entirety. NK cells that may be used according to these assays are publicly available (e.g.,</p>	<p>response, and suppressing a T cell-mediated immune response. Additional highly preferred indications include inflammation and inflammatory disorders. An additional highly preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease"). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Preferred indications include neoplasms and cancers, such as, for example, leukemia, lymphoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications also</p>
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				through the ATCC). Exemplary human NK cells that may be used according to these assays include the NK-YT cell line, which is a human natural killer cell line with cytolytic and cytotoxic activity.	include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, asthma and allergy.
31	HCWLD74	335	Activation of transcription through serum response element in immune cells (such as natural killer cells).	Assays for the activation of transcription through the Serum Response Element (SRE) are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate serum response factors and modulate the expression of genes involved	A preferred embodiment of the invention includes a method for inhibiting (e.g., reducing) TNF alpha production. An alternative highly preferred embodiment of the invention includes a method for stimulating (e.g., increasing) TNF alpha production. Preferred indications include blood disorders (e.g., as described below under "Immune

				<p>in growth and upregulate the function of growth-related genes in many cell types. Exemplary assays for transcription through the SRE that may be used or routinely modified to test SRE activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Benson et al., J Immunol 153(9):3862-3873 (1994); and Black et al., Virus Genes 12(2):105-117 (1997), the content of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary T cells that may be used according to these assays include the NK-YT cell line,</p>	<p>Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"), Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional highly preferred indications include inflammation and inflammatory disorders, and treating joint damage in patients with rheumatoid arthritis. An additional highly preferred indication is sepsis. Highly preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Additionally, highly preferred indications include neoplasms and</p>
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				<p>which is a human natural killer cell line with cytolytic and cytotoxic activity.</p>	<p>cancers, such as, for example, leukemia, lymphoma, melanoma, glioma (e.g., malignant glioma), solid tumors, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis,</p>
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					meningitis, Lyme Disease, cardiac reperfusion injury, and asthma and allergy. An additional preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease").
31	HCWLD74	335	SEAP in NK16/STAT6		
31	HCWLD74	335	Activation of transcription through GAS response element in immune cells (such as T-cells).	Assays for the activation of transcription through the Gamma Interferon Activation Site (GAS) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate STAT transcription factors and modulate gene expression involved in a wide variety of cell functions. Exemplary assays for transcription through the GAS response element that may be used or routinely modified to test GAS-response element activity of polypeptides of the	Highly preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, for example, leukemia, lymphoma (e.g., T cell lymphoma, Burkitt's lymphoma, non-Hodgkins lymphoma, Hodgkin's disease), melanoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for

				<p>invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Matikainen et al., Blood 93(6):1980-1991 (1999); and Hentinen et al., J Immunol 155(10):4582-4587 (1995), the contents of each of which are herein incorporated by reference in its entirety. Exemplary human T cells, such as the SUPT cell line, that may be used according to these assays are publicly available (e.g., through the ATCC).</p>	<p>example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional preferred indications include inflammation and inflammatory disorders. Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"), and infection (e.g., viral infections, tuberculosis, infections associated with chronic granulomatous disease and malignant osteoporosis, and/or an infectious disease as described below under "Infectious</p>
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					<p>Disease"). An additional preferred indication is idiopathic pulmonary fibrosis. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, and asthma and allergy.</p>
32	HDHEB60	336	<p>Activation of transcription through cAMP response element (CRE) in pre-adipocytes.</p>	<p>Assays for the activation of transcription through the cAMP response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to increase cAMP,</p>	<p>A highly preferred indication is obesity and/or complications associated with obesity. Additional highly preferred indications include weight loss or alternatively, weight gain. An additional highly preferred indication is diabetes mellitus. An additional highly preferred indication is a complication</p>

				<p>regulate CREB transcription factors, and modulate expression of genes involved in a wide variety of cell functions. For example, a 3T3-L1/CRE reporter assay may be used to identify factors that activate the cAMP signaling pathway. CREB plays a major role in adipogenesis, and is involved in differentiation into adipocytes. CRE contains the binding sequence for the transcription factor CREB (CRE binding protein). Exemplary assays for transcription through the cAMP response element that may be used or routinely modified to test cAMP-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn</p>	<p>associated with diabetes (e.g., diabetic retinopathy, diabetic nephropathy, kidney disease (e.g., renal failure, nephropathy and/or other diseases and disorders as described in the "Renal Disorders" section below), diabetic neuropathy, nerve disease and nerve damage (e.g., due to diabetic neuropathy), blood vessel blockage, heart disease, stroke, impotence (e.g., due to diabetic neuropathy or blood vessel blockage), seizures, mental confusion, drowsiness, nonketotic hyperglycemic-hyperosmolar coma, cardiovascular disease (e.g., heart disease, atherosclerosis, microvascular disease, hypertension, stroke, and other diseases and disorders as described in the "Cardiovascular Disorders" section below), dyslipidemia, endocrine disorders (as described in the "Endocrine Disorders" section below),</p>
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				<p>et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Reusch et al., Mol Cell Biol 20(3):1008-1020 (2000); and Klemm et al., J Biol Chem 273:917-923 (1998), the contents of each of which are herein incorporated by reference in its entirety. Pre-adipocytes that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary mouse adipocyte cells that may be used according to these assays include 3T3-L1 cells. 3T3-L1 is an adherent mouse preadipocyte cell line that is a continuous substrain of 3T3 fibroblast cells developed through clonal isolation and undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation conditions known in the art.</p>	<p>neuropathy, vision impairment (e.g., diabetic retinopathy and blindness), ulcers and impaired wound healing, and infection (e.g., infectious diseases and disorders as described in the "Infectious Diseases" section below, especially of the urinary tract and skin), carpal tunnel syndrome and Dupuytren's contracture). Additional highly preferred indications are complications associated with insulin resistance.</p>
32	HDHEB60	336	Myoblast cell proliferation	Assays for muscle cell proliferation are well known in the art and may be used or	Highly preferred indications include diabetes, myopathy, muscle cell atrophy, cancers of

				<p>routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit myoblast cell proliferation. Exemplary assays for myoblast cell proliferation that may be used or routinely modified to test activity of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) include, for example, assays disclosed in: Soeta, C., et al. "Possible role for the c-ski gene in the proliferation of myogenic cells in regenerating skeletal muscles of rats" Dev Growth Differ Apr;43(2):155-64 (2001); Ewton DZ, et al., "IGF binding proteins-4, -5 and -6 may play specialized roles during L6 myoblast proliferation and differentiation" J Endocrinol Mar;144(3):539-53 (1995); and, Pampusch MS, et al., "Effect of transforming</p>	<p>muscle (such as, rhabdomyoma, and rhabdosarcoma), cardiovascular disorders (such as congestive heart failure, cachexia, myxomas, fibromas, congenital cardiovascular abnormalities, heart disease, cardiac arrest, heart valve disease, vascular disease, and also as described below under "Cardiovascular Disorders"), stimulating myoblast proliferation, and inhibiting myoblast proliferation.</p>
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					growth factor beta on proliferation of L6 and embryonic porcine myogenic cells" J Cell Physiol Jun; 143(3):524-8 (1990); the contents of each of which are herein incorporated by reference in their entirety. Exemplary myoblast cells that may be used according to these assays include the rat myoblast L6 cell line. Rat myoblast L6 cells are an adherent rat myoblast cell line, isolated from primary cultures of rat thigh muscle, that fuse to form multinucleated myotubes and striated fibers after culture in differentiation media.	
32	HDHEB60	336	Production of VCAM in endothelial cells (such as human umbilical vein endothelial cells (HUVEC))	Assays for measuring expression of VCAM are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate VCAM expression. For example, FMAT may be used to measure	Highly preferred indications include inflammation (acute and chronic), restnosis, atherosclerosis, asthma and allergy. Highly preferred indications include inflammation and inflammatory disorders, immunological disorders, neoplastic disorders (e.g. cancer/tumorigenesis), and	

				<p>the upregulation of cell surface VCAM-1 expression in endothelial cells. Endothelial cells are cells that line blood vessels, and are involved in functions that include, but are not limited to, angiogenesis, vascular permeability, vascular tone, and immune cell extravasation. Exemplary endothelial cells that may be used according to these assays include human umbilical vein endothelial cells (HUVEC), which are available from commercial sources. The expression of VCAM (CD106), a membrane-associated protein, can be upregulated by cytokines or other factors, and contributes to the extravasation of lymphocytes, leucocytes and other immune cells from blood vessels; thus VCAM expression plays a role in promoting immune and inflammatory responses.</p>	<p>cardiovascular disorders (such as described below under "Immune Activity", "Blood-Related Disorders", "Hyperproliferative Disorders" and/or "Cardiovascular Disorders"). Highly preferred indications include neoplasms and cancers such as, for example, leukemia, lymphoma, melanoma, renal cell carcinoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia.</p>
32	HDHEB60	336	Activation of transcription	Assays for the activation of transcription through the	Highly preferred indications include blood disorders (e.g.,

			through NFAT response element in immune cells (such as natural killer cells).	<p>Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Aramburu et al., J Exp Med</p>	<p>as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional highly preferred indications include inflammation and inflammatory disorders. An additional highly preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease"). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Preferred</p>
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32	HDHEB60	336	<p>Activation of transcription through NFKB response element in immune cells (such as natural killer cells).</p>	<p>Assays for the activation of transcription through the NFKB response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription through the NFKB response element that may be used or routinely modified to test NFKB-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Valle Blazquez et al, Immunology</p>	<p>Highly preferred indications include inflammation and inflammatory disorders. Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), and immunodeficiencies (e.g., as described below). An additional highly preferred indication is infection (e.g., AIDS, and/or an infectious disease as described below under "Infectious Disease"). Highly preferred indications include neoplastic diseases (e.g., melanoma, leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms</p>
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				<p>90(3):455-460 (1997); Aramburau et al., J Exp Med 82(3):801-810 (1995); and Fraser et al., 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. NK cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary NK cells that may be used according to these assays include the NK-YT cell line, which is a human natural killer cell line with cytolytic and cytotoxic activity.</p>	<p>and cancers, such as, for example, melanoma, renal cell carcinoma, leukemia, lymphoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications also include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, suppression of immune reactions to transplanted organs, asthma and allergy.</p>
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32	HDHEB60	336	SEAP in NK16/STAT6		
32	HDHEB60	336	Activation of transcription through API response element in immune cells (such as T-cells).	Assays for the activation of transcription through the API1 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate growth and other cell functions. Exemplary assays for transcription through the API1 response element that may be used or routinely modified to test API1-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1988); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Rellahan et al., J Biol Chem 272(49):30806-30811 (1997);	Preferred indications include neoplastic diseases (e.g., as described below under "Hyperproliferative Disorders"), blood disorders (e.g., as described below under "Immune Activity", "Cardiovascular Disorders", and/or "Blood-Related Disorders"), and infection (e.g., an infectious disease as described below under "Infectious Disease"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Additional highly preferred indications include inflammation and inflammatory disorders. Highly preferred indications also include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described

				<p>Chang et al., Mol Cell Biol 18(9):4986-4993 (1998); and Fraser et al., Eur J Immunol 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. Human T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is an IL-2 and IL-4 responsive suspension-culture cell line.</p>	<p>below under “Hyperproliferative Disorders”). Highly preferred indications include neoplasms and cancers, such as, leukemia, lymphoma, prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver, and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include arthritis, asthma, AIDS, allergy, anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin’s disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt’s lymphoma, granulomatous disease, inflammatory bowel disease, sepsis, psoriasis, suppression of immune reactions to transplanted organs and tissues, endocarditis, meningitis, and Lyme Disease.</p>
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32	HDHEB60	336	<p>Activation of transcription through CD28 response element in immune cells (such as T-cells).</p>	<p>Assays for the activation of transcription through the CD28 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate IL-2 expression in T cells. Exemplary assays for transcription through the CD28 response element that may be used or routinely modified to test CD28-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); McGuire and Iacobelli, J Immunol 159(3):1319-1327 (1997); Parra et al., J Immunol 166(4):2437-2443 (2001); and</p>	<p>A highly preferred embodiment of the invention includes a method for stimulating T cell proliferation. An alternative highly preferred embodiment of the invention includes a method for inhibiting T cell proliferation. A highly preferred embodiment of the invention includes a method for activating T cells. An alternative highly preferred embodiment of the invention includes a method for inhibiting the activation of and/or inactivating T cells. A highly preferred embodiment of the invention includes a method for stimulating (e.g., increasing) IL-2 production. An alternative highly preferred embodiment of the invention includes a method for inhibiting (e.g., reducing) IL-2 production. Additional highly preferred indications include inflammation and inflammatory disorders.</p>
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				<p>Butscher et al., J Biol Chem 3(1):552-560 (1998), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.</p>	<p>Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Highly preferred indications include neoplastic diseases (e.g., melanoma, renal cell carcinoma, leukemia, lymphoma, and/or as described below under “Hyperproliferative Disorders”). Highly preferred indications include neoplasms and cancers, such as, for example, melanoma (e.g., metastatic melanoma), renal cell carcinoma (e.g., metastatic renal cell carcinoma), leukemia, lymphoma (e.g., T cell lymphoma), and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other</p>
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					<p>preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. A highly preferred indication includes infection (e.g., AIDS, tuberculosis, infections associated with granulomatous disease, and osteoporosis, and/or as described below under "Infectious Disease"). A highly preferred indication is AIDS. Additional highly preferred indications include suppression of immune reactions to transplanted organs and/or tissues, uveitis, psoriasis, and tropical spastic paraparesis. Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Preferred indications also include anemia, pancytopenia, leukopenia, thrombocytopenia,</p>
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					<p>Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, asthma and allergy.</p>
32	HDHEB60	336	<p>Activation of transcription through GAS response element in immune cells (such as T-cells).</p>	<p>Assays for the activation of transcription through the Gamma Interferon Activation Site (GAS) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate STAT transcription factors and modulate gene expression involved in a wide variety of cell functions. Exemplary assays for transcription through the GAS response element that may be used or</p>	<p>Highly preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, for example, leukemia, lymphoma (e.g., T cell lymphoma, Burkitt's lymphoma, non-Hodgkins lymphoma, Hodgkin's disease), melanoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include</p>

				<p>routinely modified to test GAS-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Matikainen et al., Blood 93(6):1980-1991 (1999); and Henttinen et al., J Immunol 155(10):4582-4587 (1995), the contents of each of which are herein incorporated by reference in its entirety. Exemplary human T cells, such as the SUPT cell line, that may be used according to these assays are publicly available (e.g., through the ATCC).</p>	<p>benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional preferred indications include inflammation and inflammatory disorders. Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"), and infection (e.g., viral infections, tuberculosis, infections associated with chronic granulomatous disease and malignant</p>
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					<p>osteoporosis, and/or an infectious disease as described below under "Infectious Disease"). An additional preferred indication is idiopathic pulmonary fibrosis. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, and asthma and allergy.</p>
32	HDHEB60	336	<p>Activation of transcription through NFAT response element in immune cells (such as T-cells).</p>	<p>Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability</p>	<p>Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Highly preferred indications</p>

			<p>of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Serfling et al., Biochim Biophys Acta 1498(1):1-18 (2000); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Fraser et al., Eur J Immunol 29(3):838-844 (1999); and</p>	<p>include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional highly preferred indications include inflammation and inflammatory disorders. An additional highly preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease"). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Preferred indications include neoplasms and cancers, such as, for example, leukemia, lymphoma, and prostate, breast, lung, colon, pancreatic, esophageal,</p>
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32	HDHEB60	336	<p>Activation of transcription through NFKB response element in immune cells (such</p>	<p>Assays for the activation of transcription through the NFKB response element are well-known in the art and may be used or routinely modified</p>	<p>Yeseen et al., J Biol Chem 268(19):14285-14293 (1993), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.</p>	<p>stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications also include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, asthma and allergy.</p>	<p>Highly preferred indications include inflammation and inflammatory disorders. Highly preferred indications include blood disorders (e.g.,</p>
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			as T-cells).	<p>to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFkB transcription factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription through the NFkB response element that may be used or routinely modified to test NFkB-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Black et al., Virus Gnes 15(2):105-117 (1997); and Fraser et al., 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. T</p>	<p>as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), and immunodeficiencies (e.g., as described below). An additional highly preferred indication is infection (e.g., AIDS, and/or an infectious disease as described below under "Infectious Disease"). Highly preferred indications include neoplastic diseases (e.g., melanoma, leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, melanoma, renal cell carcinoma, leukemia, lymphoma, and prostate, breast, lung, colon, pancreatic,</p>
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				<p>cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.</p>	<p>esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications also include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, suppression of immune reactions to transplanted organs, asthma and allergy.</p>
32	HDHEB60	336	<p>Activation of transcription through STAT6 response element in</p>	<p>Assays for the activation of transcription through the Signal Transducers and Activators of Transcription</p>	<p>A highly preferred indication is allergy. Another highly preferred indication is asthma.</p>

			immune cells (such as T-cells).	<p>(STAT6) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate STAT6 transcription factors and modulate the expression of multiple genes. Exemplary assays for transcription through the STAT6 response element that may be used or routinely modified to test STAT6 response element activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Georas et al., Blood 92(12):4529-4538 (1998); Moffatt et al., Transplantation 69(7):1521-</p>	<p>Additional highly preferred indications include inflammation and inflammatory disorders. Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, melanoma, and/or as described below under "Hyperproliferative Disorders"). Preferred indications include neoplasms and cancers, such as, leukemia, lymphoma, melanoma, and prostate, breast, lung, colon, pancreatic, esophageal,</p>
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				<p>1523 (2000); Curiel et al., Eur J Immunol 27(8):1982-1987 (1997); and Masuda et al., J Biol Chem 275(38):29331-29337 (2000), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.</p>	<p>stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, and Lyme Disease. An additional preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease").</p>
	HDHMA45	337	IFN γ in Human T-		

33			cell 293T		
33	HDHMA45	337	SEAP in Jurkat/IL4 promoter		
33	HDHMA45	337	Production of IL-10 and activation of T-cells.	Assays for production of IL-10 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed	Highly preferred indications include allergy and asthma. Additional highly preferred indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response.

				therapy for asthma" Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.	
34	HDHMA72	338	Activation of transcription through NFkB response element in immune cells (such	Assays for the activation of transcription through the NFkB response element are well-known in the art and may be used or routinely modified	Highly preferred indications include inflammation and inflammatory disorders. Highly preferred indications include blood disorders (e.g.,

			<p>to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFκB transcription factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription through the NFκB response element that may be used or routinely modified to test NFκB-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Valle Blazquez et al, Immunology 90(3):455-460 (1997); Aramburau et al., J Exp Med 82(3):801-810 (1995); and Fraser et al., 29(3):838-844 (1999), the contents of each of</p>	<p>as natural killer cells).</p>	<p>to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFκB transcription factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription through the NFκB response element that may be used or routinely modified to test NFκB-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Valle Blazquez et al, Immunology 90(3):455-460 (1997); Aramburau et al., J Exp Med 82(3):801-810 (1995); and Fraser et al., 29(3):838-844 (1999), the contents of each of</p>	<p>as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), and immunodeficiencies (e.g., as described below). An additional highly preferred indication is infection (e.g., AIDS, and/or an infectious disease as described below under "Infectious Disease"). Highly preferred indications include neoplastic diseases (e.g., melanoma, leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, for example, melanoma, renal cell carcinoma, leukemia, lymphoma, and prostate, breast, lung, colon, pancreatic,</p>
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			<p>which are herein incorporated by reference in its entirety. NK cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human NK cells that may be used according to these assays include the NKL cell line, which is a human natural killer cell line established from the peripheral blood of a patient with large granular lymphocytic leukemia. This IL-2 dependent suspension culture cell line has a morphology resembling that of activated NK cells.</p>	<p>esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications also include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, suppression of immune reactions to transplanted organs, asthma and allergy.</p>
35	HDPBA28	339	<p>Stimulation of insulin secretion from pancreatic beta cells.</p>	<p>Assays for measuring secretion of insulin are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of</p> <p>A highly preferred indication is diabetes mellitus. An additional highly preferred indication is a complication associated with diabetes (e.g.,</p>

			<p>the invention (including antibodies and agonists or antagonists of the invention) to stimulate insulin secretion. For example, insulin secretion is measured by FMAT using anti-rat insulin antibodies. Insulin secretion from pancreatic beta cells is upregulated by glucose and also by certain proteins/peptides, and dysregulation is a key component in diabetes. Exemplary assays that may be used or routinely modified to test for stimulation of insulin secretion (from pancreatic cells) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Ahren, B., et al., <i>Am J Physiol</i>, 277(4 Pt 2):R959-66 (1999); Li, M., et al., <i>Endocrinology</i>, 138(9):3735-40 (1997); Kim, K.H., et al., <i>FEBS Lett</i>, 377(2):237-9 (1995); and, Miraglia S et. al., <i>Journal of</i></p>	<p>diabetic retinopathy, diabetic nephropathy, kidney disease (e.g., renal failure, nephropathy and/or other diseases and disorders as described in the "Renal Disorders" section below), diabetic neuropathy, nerve disease and nerve damage (e.g., due to diabetic neuropathy), blood vessel blockage, heart disease, stroke, impotence (e.g., due to diabetic neuropathy or blood vessel blockage), seizures, mental confusion, drowsiness, nonketotic hyperglycemic-hypermolar coma, cardiovascular disease (e.g., heart disease, atherosclerosis, microvascular disease, hypertension, stroke, and other diseases and disorders as described in the "Cardiovascular Disorders" section below), dyslipidemia, endocrine disorders (as described in the "Endocrine Disorders" section below), neuropathy, vision impairment</p>
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				<p>Biomolecular Screening, 4:193-204 (1999), the contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include rat INS-1 cells. INS-1 cells are a semi-adherent cell line established from cells isolated from an X-ray induced rat transplantable insulinoma. These cells retain characteristics typical of native pancreatic beta cells including glucose inducible insulin secretion. References: Asfari et al. Endocrinology 1992 130:167.</p>	<p>(e.g., diabetic retinopathy and blindness), ulcers and impaired wound healing, and infection (e.g., infectious diseases and disorders as described in the "Infectious Diseases" section below, especially of the urinary tract and skin), carpal tunnel syndrome and Dupuytren's contracture). An additional highly preferred indication is obesity and/or complications associated with obesity. Additional highly preferred indications include weight loss or alternatively, weight gain. Additional highly preferred indications are complications associated with insulin resistance.</p>
35	HDPBA28	339	Production of IL-10 and activation of T-cells.	<p>Assays for production of IL-10 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention</p>	<p>Highly preferred indications include allergy and asthma. Additional highly preferred indications include immune and hematopoietic disorders (e.g., as described below under</p>

				<p>(including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL10</p>	<p>"Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response.</p>
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36	HDPCO25	340	Regulation of viability and proliferation of pancreatic beta cells.	secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.	A highly preferred indication is diabetes mellitus. An additional highly preferred indication is a complication associated with diabetes (e.g., diabetic retinopathy, diabetic nephropathy, kidney disease (e.g., renal failure, nephropathy and/or other diseases and disorders as described in the "Renal Disorders" section below), diabetic neuropathy, nerve disease and nerve damage
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				<p>number of viable cells in culture based on quantitation of the ATP present which signals the presence of metabolically active cells. Exemplary assays that may be used or routinely modified to test regulation of viability and proliferation of pancreatic beta cells by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Ohtani KI, et al., Endocrinology, 139(1):172-8 (1998); Krauthaim A, et al., Exp Clin Endocrinol Diabetes, 107 (1):29-34 (1999), the contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include HIT T15 Cells. HIT T15 are an adherent</p>	<p>(e.g., due to diabetic neuropathy), blood vessel blockage, heart disease, stroke, impotence (e.g., due to diabetic neuropathy or blood vessel blockage), seizures, mental confusion, drowsiness, nonketotic hyperglycemic-hyperosmolar coma, cardiovascular disease (e.g., heart disease, atherosclerosis, microvascular disease, hypertension, stroke, and other diseases and disorders as described in the "Cardiovascular Disorders" section below), dyslipidemia, endocrine disorders (as described in the "Endocrine Disorders" section below), neuropathy, vision impairment (e.g., diabetic retinopathy and blindness), ulcers and impaired wound healing, and infection (e.g., infectious diseases and disorders as described in the "Infectious Diseases" section below, especially of the urinary tract and skin), carpal tunnel syndrome and</p>
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				epithelial cell line established from Syrian hamster islet cells transformed with SV40. These cells express glucagon, somatostatin, and glucocorticoid receptors. The cells secrete insulin, which is stimulated by glucose and glucagon and suppressed by somatostatin or glucocorticoids. ATTC# CRL-1777 Refs: Lord and Ashcroft. Biochem. J. 219: 547-551; Santerre et al. Proc. Natl. Acad. Sci. USA 78: 4339-4343, 1981.	Dupuytren's contracture). An additional highly preferred indication is obesity and/or complications associated with obesity. Additional highly preferred indications include weight loss or alternatively, weight gain. Additional highly preferred indications are complications associated with insulin resistance.
36	HDPCO25	340	Activation of transcription through NFKB response element in immune cells (such as T-cells).	Assays for the activation of transcription through the NFKB response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription factors and modulate expression of immunomodulatory genes. Exemplary assays for	Highly preferred indications include inflammation and inflammatory disorders. Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as

			transcription through the NFKB response element that may be used or routinely modified to test NFKB-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Black et al., Virus Gnes 15(2):105-117 (1997); and Fraser et al., 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4	described below), and immunodeficiencies (e.g., as described below). An additional highly preferred indication is infection (e.g., AIDS, and/or an infectious disease as described below under "Infectious Disease"). Highly preferred indications include neoplastic diseases (e.g., melanoma, leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, melanoma, renal cell carcinoma, leukemia, lymphoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications also
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				responsive T cells.	include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, suppression of immune reactions to transplanted organs, asthma and allergy.
37	HDPCY37	341	Activation of transcription through cAMP response element (CRE) in pre-adipocytes.	Assays for the activation of transcription through the cAMP response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to increase cAMP, regulate CREB transcription factors, and modulate expression of genes involved	A highly preferred indication is obesity and/or complications associated with obesity. Additional highly preferred indications include weight loss or alternatively, weight gain. An additional highly preferred indication is diabetes mellitus. An additional highly preferred indication is a complication associated with diabetes (e.g., diabetic retinopathy, diabetic nephropathy, kidney disease

				<p>in a wide variety of cell functions. For example, a 3T3-L1/CRE reporter assay may be used to identify factors that activate the cAMP signaling pathway. CREB plays a major role in adipogenesis, and is involved in differentiation into adipocytes. CRE contains the binding sequence for the transcription factor CREB (CRE binding protein). Exemplary assays for transcription through the cAMP response element that may be used or routinely modified to test cAMP-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Reusch et al., Mol Cell Biol</p>	<p>(e.g., renal failure, nephropathy and/or other diseases and disorders as described in the "Renal Disorders" section below), diabetic neuropathy, nerve disease and nerve damage (e.g., due to diabetic neuropathy), blood vessel blockage, heart disease, stroke, impotence (e.g., due to diabetic neuropathy or blood vessel blockage), seizures, mental confusion, drowsiness, nonketotic hyperglycemic-hypermolar coma, cardiovascular disease (e.g., heart disease, atherosclerosis, microvascular disease, hypertension, stroke, and other diseases and disorders as described in the "Cardiovascular Disorders" section below), dyslipidemia, endocrine disorders (as described in the "Endocrine Disorders" section below), neuropathy, vision impairment (e.g., diabetic retinopathy and blindness), ulcers and impaired</p>
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37	HDPCY37	341	<p>Activation of transcription through NFkB response element in immune cells (such as T-cells).</p>	<p>Assays for the activation of transcription through the NFkB response element are well-known in the art and may be used or routinely modified to assess the ability of</p>	<p>20(3):1008-1020 (2000); and Klemm et al., J Biol Chem 273:917-923 (1998), the contents of each of which are herein incorporated by reference in its entirety. Pre-adipocytes that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary mouse adipocyte cells that may be used according to these assays include 3T3-L1 cells. 3T3-L1 is an adherent mouse preadipocyte cell line that is a continuous substrain of 3T3 fibroblast cells developed through clonal isolation and undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation conditions known in the art.</p>	<p>wound healing, and infection (e.g., infectious diseases and disorders as described in the "Infectious Diseases" section below, especially of the urinary tract and skin), carpal tunnel syndrome and Dupuytren's contracture). Additional highly preferred indications are complications associated with insulin resistance.</p>	<p>Highly preferred indications include inflammation and inflammatory disorders. Highly preferred indications include blood disorders (e.g., as described below under</p>
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				<p>polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFkB transcription factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription through the NFkB response element that may be used or routinely modified to test NFkB-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Black et al., Virus Gnes 15(2):105-117 (1997); and Fraser et al., 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used</p>	<p>"Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), and immunodeficiencies (e.g., as described below). An additional highly preferred indication is infection (e.g., AIDS, and/or an infectious disease as described below under "Infectious Disease"). Highly preferred indications include neoplastic diseases (e.g., melanoma, leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, melanoma, renal cell carcinoma, leukemia, lymphoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain,</p>
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				<p>according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.</p>	<p>liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications also include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, suppression of immune reactions to transplanted organs, asthma and allergy.</p>
37	HDPCY37	341	<p>Production of IL-10 and activation of T-cells.</p>	<p>Assays for production of IL-10 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of</p>	<p>Highly preferred indications include allergy and asthma. Additional highly preferred indications include immune and hematopoietic disorders</p>

				<p>polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays</p>	<p>(e.g., as described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response.</p>
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38	HDPHI51	342		include Th2 cells. IL10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.	
			Regulation of transcription through the FAS promoter element in hepatocytes	Assays for the regulation of transcription through the FAS promoter element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to activate the FAS promoter element in a reporter construct and to regulate transcription of FAS, a key	A highly preferred indication is diabetes mellitus. An additional highly preferred indication is a complication associated with diabetes (e.g., diabetic retinopathy, diabetic nephropathy, kidney disease (e.g., renal failure, nephropathy and/or other diseases and disorders as described in the "Renal Disorders" section below), diabetic neuropathy, nerve

			<p>enzyme for lipogenesis. FAS promoter is regulated by many transcription factors including SREBP. Insulin increases FAS gene transcription in livers of diabetic mice. This stimulation of transcription is also somewhat glucose dependent. Exemplary assays that may be used or routinely modified to test for FAS promoter element activity (in hepatocytes) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Xiong, S., et al., Proc Natl Acad Sci U.S.A., 97(8):3948-53 (2000); Roder, K., et al., Eur J Biochem, 260(3):743-51 (1999); Oskouian B, et al., Biochem J, 317 (Pt 1):257-65 (1996); Berger, et al., Gene 66:1-10 (1988); and, Cullen, B., et al., Methods in Enzymol. 216:362-368 (1992), the contents of each of which is herein incorporated by reference in its entirety.</p>	<p>disease and nerve damage (e.g., due to diabetic neuropathy), blood vessel blockage, heart disease, stroke, impotence (e.g., due to diabetic neuropathy or blood vessel blockage), seizures, mental confusion, drowsiness, nonketotic hyperglycemic-hyperosmolar coma, cardiovascular disease (e.g., heart disease, atherosclerosis, microvascular disease, hypertension, stroke, and other diseases and disorders as described in the "Cardiovascular Disorders" section below), dyslipidemia, endocrine disorders (as described in the "Endocrine Disorders" section below), neuropathy, vision impairment (e.g., diabetic retinopathy and blindness), ulcers and impaired wound healing, and infection (e.g., infectious diseases and disorders as described in the "Infectious Diseases" section below, especially of the urinary tract and skin), carpal</p>
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				<p>Hepatocytes that may be used according to these assays, such as H4IIE cells, are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary hepatocytes that may be used according to these assays include rat liver hepatoma cell line(s) inducible with glucocorticoids, insulin, or cAMP derivatives.</p>	<p>tunnel syndrome and Dupuytren's contracture). An additional highly preferred indication is obesity and/or complications associated with obesity. Additional highly preferred indications include weight loss or alternatively, weight gain. Additional highly preferred indications are complications associated with insulin resistance.</p>
38	HDPH151	342	<p>Activation of transcription through STAT6 response element in immune cells (such as T-cells).</p>	<p>Assays for the activation of transcription through the Signal Transducers and Activators of Transcription (STAT6) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate STAT6 transcription factors and modulate the expression of multiple genes. Exemplary assays for transcription through the STAT6 response element that may be used or</p>	<p>A highly preferred indication is allergy. Another highly preferred indication is asthma. Additional highly preferred indications include inflammation and inflammatory disorders. Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple</p>

				<p>routinely modified to test STAT6 response element activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Georas et al., Blood 92(12):4529-4538 (1998); Moffatt et al., Transplantation 69(7):1521-1523 (2000); Curiel et al., Eur J Immunol 27(8):1982-1987 (1997); and Masuda et al., J Biol Chem 275(38):29331-29337 (2000), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary T cells that may be used according to these assays include the SUPT cell line,</p>	<p>sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, melanoma, and/or as described below under "Hyperproliferative Disorders"). Preferred indications include neoplasms and cancers, such as, leukemia, lymphoma, melanoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma,</p>
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				which is a suspension culture of IL-2 and IL-4 responsive T cells.	arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, and Lyme Disease. An additional preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease").
39	HDPND46	343	Activation of Adipocyte PI3 Kinase Signalling Pathway	Kinase assay. Kinase assays, for example an GSK-3 assays, for PI3 kinase signal transduction that regulate glucose metabolism and cell survival are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit glucose metabolism and cell survival. Exemplary assays for PI3	<p>A highly preferred embodiment of the invention includes a method for increasing adipocyte survival</p> <p>An alternative highly preferred embodiment of the invention includes a method for decreasing adipocyte survival.</p> <p>A preferred embodiment of the invention includes a method for stimulating adipocyte proliferation. An alternative highly preferred embodiment of the invention includes a method for inhibiting</p>

				<p>kinase activity that may be used or routinely modified to test PI3 kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Nikoulina et al., Diabetes 49(2):263-271 (2000); and Schreyer et al., Diabetes 48(8):1662-1666 (1999), the contents of each of which are herein incorporated by reference in its entirety. Mouse adipocyte cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary mouse adipocyte cells that may be used according to these assays include 3T3-L1 cells. 3T3-L1 is an adherent mouse preadipocyte cell line that is a continuous substrain of 3T3 fibroblast cells developed through clonal isolation and undergo a pre-adipocyte to</p>	<p>adipocyte proliferation. A preferred embodiment of the invention includes a method for stimulating adipocyte differentiation. An alternative highly preferred embodiment of the invention includes a method for inhibiting adipocyte differentiation. Highly preferred indications include endocrine disorders (e.g., as described below under "Endocrine Disorders"). Preferred indications include neoplastic diseases (e.g., lipomas, liposarcomas, and/or as described below under "Hyperproliferative Disorders"), blood disorders (e.g., hypertension, congestive heart failure, blood vessel blockage, heart disease, stroke, impotence and/or as described below under "Immune Activity", "Cardiovascular Disorders", and/or "Blood-Related Disorders"), immune disorders (e.g., as described below under "Immune Activity"), neural disorders</p>
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				adipose-like conversion under appropriate differentiation conditions known in the art.	(e.g., as described below under "Neural Activity and Neurological Diseases"), and infection (e.g., as described below under "Infectious Disease"). A highly preferred indication is diabetes mellitus. An additional highly preferred indication is a complication associated with diabetes (e.g., diabetic retinopathy, diabetic nephropathy, kidney disease (e.g., renal failure, nephropathy and/or other diseases and disorders as described in the "Renal Disorders" section below), diabetic neuropathy, nerve disease and nerve damage (e.g., due to diabetic neuropathy), blood vessel blockage, heart disease, stroke, impotence (e.g., due to diabetic neuropathy or blood vessel blockage), seizures, mental confusion, drowsiness, nonketotic hyperglycemic-hyperosmolar coma, cardiovascular disease (e.g.,
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					heart disease, atherosclerosis, microvascular disease, hypertension, stroke, and other diseases and disorders as described in the "Cardiovascular Disorders" section below), dyslipidemia, endocrine disorders (as described in the "Endocrine Disorders" section below), neuropathy, vision impairment (e.g., diabetic retinopathy and blindness), ulcers and impaired wound healing, infection (e.g., infectious diseases and disorders as described in the "Infectious Diseases" section below, especially of the urinary tract and skin), carpal tunnel syndrome and Dupuytren's contracture). An additional highly preferred indication is obesity and/or complications associated with obesity. Additional highly preferred indications include weight loss or alternatively, weight gain. Additional highly preferred indications are complications associated with

					<p>insulin resistance.</p> <p>Additional highly preferred indications are disorders of the musculoskeletal systems including myopathies, muscular dystrophy, and/or as described herein.</p> <p>Additional highly preferred indications include, hypertension, coronary artery disease, dyslipidemia, gallstones, osteoarthritis, degenerative arthritis, eating disorders, fibrosis, cachexia, and kidney diseases or disorders. Highly preferred indications include neoplasms and cancer, such as, lipoma, liposarcoma, lymphoma, leukemia and breast, colon, and kidney cancer. Additional highly preferred indications include melanoma, prostate, lung, pancreatic, esophageal, stomach, brain, liver, and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia,</p>
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39	HDPND46	343	Production of IL-4	<p>IL-4 FMAT. Assays for immunomodulatory proteins secreted by TH2 cells that stimulate B cells, T cells, macrophages and mast cells and promote polarization of CD4+ cells into TH2 cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation, stimulate immune cells, modulate immune cell polarization, and/or mediate humoral or cell-mediated immunity. Exemplary assays that test for immunomodulatory proteins evaluate the production of cytokines, such as IL-4, and the stimulation of immune cells, such as B cells, T cells, macrophages and mast cells. Such assays that may be used or routinely modified to test immunomodulatory activity of</p>	<p>metaplasia, and/or dysplasia.</p> <p>A highly preferred embodiment of the invention includes a method for stimulating (e.g., increasing) IL-4 production. An alternative highly preferred embodiment of the invention includes a method for inhibiting (e.g., reducing) IL-4 production. A highly preferred indication includes asthma. A highly preferred indication includes allergy. A highly preferred indication includes rhinitis. Additional highly preferred indications include inflammation and inflammatory disorders. Highly preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, melanoma, and/or as described below under “Hyperproliferative Disorders”). Preferred indications include neoplasms and cancers, such as, for example, leukemia, lymphoma, melanoma, and prostate,</p>
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				<p>polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204 (1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); Gonzalez et al., J Clin Lab Anal 8(5):277-283 (1194); Yssel et al., Res Immunol 144(8):610-616 (1993); Bagley et al., Nat Immunol 1(3):257-261 (2000); and van der Graaff et al., Rheumatology (Oxford) 38(3):214-220 (1999), the contents of each of which are herein incorporated by reference in its entirety.</p> <p>Human T cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human T cells are primary human lymphocytes that mature in the thymus and express a T cell receptor and CD3, CD4, or CD8. These</p>	<p>breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma,</p>
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				cells mediate humoral or cell-mediated immunity and may be preactivated to enhance responsiveness to immunomodulatory factors.	arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, and Lyme Disease. An additional preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease").
39	HDPND46	343	Production of IL-8 by endothelial cells (such as Human Umbilical Cord Endothelial Cells).	Assays measuring production of IL-8 are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate production and/or secretion of IL-8. For example, FMAT may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or	Highly preferred indications include immunological and inflammatory disorders (e.g., such as allergy, asthma, leukemia, etc. and as described below under "Immune Activity", and "Blood-Related Disorders"). Highly preferred indications also include autoimmune disorders (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), neoplastic disorders (e.g.,

				antagonists of the invention) to regulate production and/or secretion of IL-8 from endothelial cells (such as human umbilical vein endothelial cells (HUVEC)). HUVECs are endothelial cells which line venous blood vessels, and are involved in functions that include, but are not limited to, angiogenesis, vascular permeability, vascular tone, and immune cell extravasation. Endothelial cells play a pivotal role in the initiation and perpetuation of inflammation and secretion of IL-8 may play an important role in recruitment and activation of immune cells such as neutrophils, macrophages, and lymphocytes.	organ cancers such as lung, liver, colon cancer, and/or as described below under "Hyperproliferative Disorders"), and cardiovascular disorders (e.g. such as described below under "Cardiovascular Disorders"). Preferred indications include thrombosis, bacteremia and sepsis syndrome and consequent complications (such as acute respiratory distress syndrome and systemic ischemia-reperfusion resulting from septic shock), restenosis and atherosclerosis.
40	HDPOH06	344	Production of ICAM-1	Assays for measuring expression of ICAM-1 are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and	Preferred embodiments of the invention include using polypeptides of the invention (or antibodies, agonists, or antagonists thereof) in detection, diagnosis, prevention, and/or treatment of

				agonists or antagonists of the invention) to regulate ICAM-1 expression. Exemplary assays that may be used or routinely modified to measure ICAM-1 expression include assays disclosed in: Takacs P, et al, FASEB J, 15(2):279-281 (2001); and, Miyamoto K, et al., Am J Pathol, 156(5):1733-1739 (2000), the contents of each of which is herein incorporated by reference in its entirety. Cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary cells that may be used according to these assays include microvascular endothelial cells (MVEC).	Inflammation, Vascular Disease, Atherosclerosis, Restenosis, and Stroke
40	HDPOH06	344	Production of IL-10 and activation of T-cells.	Assays for production of IL-10 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the	Highly preferred indications include allergy and asthma. Additional highly preferred indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"),

				<p>invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL10 secreted from Th2 cells may be measured as a marker of Th2</p>	<p>autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response.</p>
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41	HDPSP54	345	Activation of Endothelial Cell JNK Signaling Pathway.	<p>cell activation. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.</p> <p>Kinase assay. JNK kinase assays for signal transduction that regulate cell proliferation, activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and apoptosis. Exemplary assays for JNK kinase activity that may be used or routinely modified to</p>	<p>A highly preferred embodiment of the invention includes a method for stimulating endothelial cell growth. An alternative highly preferred embodiment of the invention includes a method for inhibiting endothelial cell growth. A highly preferred embodiment of the invention includes a method for stimulating endothelial cell proliferation. An alternative highly preferred embodiment of the invention includes a method for inhibiting</p>
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			test JNK kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. Endothelial cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary endothelial cells that may be used according to these assays include human umbilical vein endothelial cells (HUVEC), which are endothelial cells which line venous blood vessels, and are involved in functions that	endothelial cell proliferation. A highly preferred embodiment of the invention includes a method for stimulating apoptosis of endothelial cells. An alternative highly preferred embodiment of the invention includes a method for inhibiting apoptosis of endothelial cells. A highly preferred embodiment of the invention includes a method for stimulating endothelial cell activation. An alternative highly preferred embodiment of the invention includes a method for inhibiting the activation of and/or inactivating endothelial cells. A highly preferred embodiment of the invention includes a method for stimulating angiogenesis. An alternative highly preferred embodiment of the invention includes a method for inhibiting angiogenesis. A highly preferred embodiment of the invention includes a
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				<p>include, but are not limited to, angiogenesis, vascular permeability, vascular tone, and immune cell extravasation.</p> <p>method for reducing cardiac hypertrophy. An alternative highly preferred embodiment of the invention include a method for inducing cardiac hypertrophy. Highly preferred indications include neoplastic diseases (e.g., as described below under “Hyperproliferative Disorders”), and disorders of the cardiovascular system (e.g., heart disease, congestive heart failure, hypertension, aortic stenosis, cardiomyopathy, valvular regurgitation, left ventricular dysfunction, atherosclerosis and atherosclerotic vascular disease, diabetic nephropathy, intracardiac shunt, cardiac hypertrophy, myocardial infarction, chronic hemodynamic overload, and/or as described below under “Cardiovascular Disorders”). Highly preferred indications include cardiovascular, endothelial and/or angiogenic disorders (e.g., systemic</p>
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					<p>disorders that affect vessels such as diabetes mellitus, as well as diseases of the vessels themselves, such as of the arteries, capillaries, veins and/or lymphatics). Highly preferred are indications that stimulate angiogenesis and/or cardiovascularization. Highly preferred are indications that inhibit angiogenesis and/or cardiovascularization.</p> <p>Highly preferred indications include antiangiogenic activity to treat solid tumors, leukemias, and Kaposi's sarcoma, and retinal disorders.</p> <p>Highly preferred indications include neoplasms and cancer, such as, Kaposi's sarcoma, hemangioma (capillary and cavernous), glomus tumors, telangiectasia, bacillary angiomatosis, hemangioendothelioma, angiosarcoma, haemangiopericytoma, lymphangioma, lymphangiosarcoma. Highly preferred indications also</p>
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					include cancers such as, prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver, and urinary cancer. Preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Highly preferred indications also include arterial disease, such as, atherosclerosis, hypertension, coronary artery disease, inflammatory vasculitides, Reynaud"s disease and Reynaud"s phenomenon, aneurysms, restenosis; venous and lymphatic disorders such as thrombophlebitis, lymphangitis, and lymphedema; and other vascular disorders such as peripheral vascular disease, and cancer. Highly preferred indications also include trauma such as wounds, burns, and injured tissue (e.g., vascular injury
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					<p>such as, injury resulting from balloon angioplasty, and atherosclerotic lesions), implant fixation, scarring, ischemia reperfusion injury, rheumatoid arthritis, cerebrovascular disease, renal diseases such as acute renal failure, and osteoporosis. Additional highly preferred indications include stroke, graft rejection, diabetic or other retinopathies, thrombotic and coagulative disorders, vasculitis, lymph angiogenesis, sexual disorders, age-related macular degeneration, and treatment /prevention of endometriosis and related conditions. Additional highly preferred indications include fibromas, heart disease, cardiac arrest, heart valve disease, and vascular disease. Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or</p>
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41	HDPSP54	345	Regulation of apoptosis in pancreatic beta cells.	Caspase Apoptosis. Assays for caspase apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote caspase protease-mediated apoptosis. Apoptosis in pancreatic beta is associated with induction and progression of diabetes.	<p>"Cardiovascular Disorders"). Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Additional preferred indications include inflammation and inflammatory disorders (such as acute and chronic inflammatory diseases, e.g., inflammatory bowel disease and Crohn's disease), and pain management.</p> <p>A highly preferred indication is diabetes mellitus. An additional highly preferred indication is a complication associated with diabetes (e.g., diabetic retinopathy, diabetic nephropathy, kidney disease (e.g., renal failure, nephropathy and/or other diseases and disorders as described in the "Renal Disorders" section below), diabetic neuropathy, nerve</p>
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				<p>Exemplary assays for caspase apoptosis that may be used or routinely modified to test caspase apoptosis activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in: Loweth, AC, et al., FEBS Lett, 400(3):285-8 (1997); Saini, KS, et al., Biochem Mol Biol Int, 39(6):1229-36 (1996); Krauthaim, A., et al., Br J Pharmacol, 129(4):687-94 (2000); Chandra J, et al., Diabetes, 50 Suppl 1:S44-7 (2001); Suk K, et al., J Immunol, 166(7):4481-9 (2001); Tejedo J, et al., FEBS Lett, 459(2):238-43 (1999); Zhang, S., et al., FEBS Lett, 455(3):315-20 (1999); Lee et al., FEBS Lett 485(2-3): 122-126 (2000); Nor et al., J Vasc Res 37(3): 209-218 (2000); and Karsan and Harlan, J Atheroscler Thromb 3(2): 75-80 (1996); the contents of each of which are herein</p>	<p>disease and nerve damage (e.g., due to diabetic neuropathy), blood vessel blockage, heart disease, stroke, impotence (e.g., due to diabetic neuropathy or blood vessel blockage), seizures, mental confusion, drowsiness, nonketotic hyperglycemic-hypermolar coma, cardiovascular disease (e.g., heart disease, atherosclerosis, microvascular disease, hypertension, stroke, and other diseases and disorders as described in the "Cardiovascular Disorders" section below), dyslipidemia, endocrine disorders (as described in the "Endocrine Disorders" section below), neuropathy, vision impairment (e.g., diabetic retinopathy and blindness), ulcers and impaired wound healing, and infection (e.g., infectious diseases and disorders as described in the "Infectious Diseases" section below, especially of the urinary tract and skin), carpal</p>
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				<p>incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include RIN-m. RIN-m is a rat adherent pancreatic beta cell insulinoma cell line derived from a radiation induced transplantable rat islet cell tumor. The cells produce and secrete islet polypeptide hormones, and produce insulin, somatostatin, and possibly glucagon. ATTC: #CRL-2057 Chick et al. Proc. Natl. Acad. Sci. 1977 74:628; AF et al. Proc. Natl. Acad. Sci. 1980 77:3519.</p>	<p>tunnel syndrome and Dupuytren's contracture). An additional highly preferred indication is obesity and/or complications associated with obesity. Additional highly preferred indications include weight loss or alternatively, weight gain. Additional highly preferred indications are complications associated with insulin resistance.</p>
41	HDPSP54	345	<p>Production of IL-10 and activation of T-cells.</p>	<p>Assays for production of IL-10 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and</p>	<p>Highly preferred indications include allergy and asthma. Additional highly preferred indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and</p>

				agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL10 secreted from Th2 cells may be	"Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response.
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				measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.	
42	HDPVH60	346	Activation of transcription through NFKB response element in immune cells (such as T-cells).	Assays for the activation of transcription through the NFKB response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription through the	Highly preferred indications include inflammation and inflammatory disorders. Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), and

				<p>NFKB response element that may be used or routinely modified to test NFKB-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Black et al., Virus Gnes 15(2):105-117 (1997); and Fraser et al., 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC).</p> <p>Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.</p>	<p>immunodeficiencies (e.g., as described below). An additional highly preferred indication is infection (e.g., AIDS, and/or an infectious disease as described below under "Infectious Disease"). Highly preferred indications include neoplastic diseases (e.g., melanoma, leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, melanoma, renal cell carcinoma, leukemia, lymphoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications also include anemia, pancytopenia,</p>
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43	HDPWN93	347	Activation of JNK Signaling Pathway in immune cells (such as eosinophils).	Kinase assay. JNK kinase assays for signal transduction that regulate cell proliferation, activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and apoptosis. Exemplary assays for JNK	leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, suppression of immune reactions to transplanted organs, asthma and allergy.
					Highly preferred indications include asthma, allergy, hypersensitivity reactions, inflammation, and inflammatory disorders. Additional highly preferred indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's

				<p>kinase activity that may be used or routinely modified to test JNK kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. Exemplary cells that may be used according to these assays include eosinophils. Eosinophils are important in the late stage of allergic reactions; they are recruited to tissues and mediate the inflammatory response of late stage allergic reaction. Moreover, exemplary assays</p>	<p>disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below). Highly preferred indications also include boosting or inhibiting immune cell proliferation. Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include boosting an eosinophil-mediated immune response, and suppressing an eosinophil-mediated immune response.</p>
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				<p>that may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate signal transduction, cell proliferation, activation, or apoptosis in eosinophils include assays disclosed and/or cited in: Zhang JP, et al., "Role of caspases in dexamethasone-induced apoptosis and activation of c-Jun NH2-terminal kinase and p38 mitogen-activated protein kinase in human eosinophils" Clin Exp Immunol; Oct;122(1):20-7 (2000); Hebestreit H, et al., "Disruption of fas receptor signaling by nitric oxide in eosinophils" J Exp Med; Feb 2;187(3):415-25 (1998); J Allergy Clin Immunol 1999 Sep;104(3 Pt 1):565-74; and, Sousa AR, et al., "In vivo resistance to corticosteroids in bronchial asthma is associated with enhanced</p>	
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				phosphorylation of JUN N-terminal kinase and failure of prednisolone to inhibit JUN N-terminal kinase phosphorylation" J Allergy Clin Immunol; Sep;104(3 Pt 1):565-74 (1999); the contents of each of which are herein incorporated by reference in its entirety.	
43	HDPWN93	347	Activation of Endothelial Cell p38 or JNK Signaling Pathway.	Kinase assay. JNK and p38 kinase assays for signal transduction that regulate cell proliferation, activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and apoptosis. Exemplary assays for JNK and p38 kinase activity that may be used or routinely modified to test JNK and p38 kinase-induced activity of polypeptides of the invention (including antibodies	A highly preferred embodiment of the invention includes a method for stimulating endothelial cell growth. An alternative highly preferred embodiment of the invention includes a method for inhibiting endothelial cell growth. A highly preferred embodiment of the invention includes a method for stimulating endothelial cell proliferation. An alternative highly preferred embodiment of the invention includes a method for inhibiting endothelial cell proliferation. A highly preferred embodiment of the invention includes a method for

			<p>and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9): 1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. Endothelial cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary endothelial cells that may be used according to these assays include human umbilical vein endothelial cells (HUVEC), which are endothelial cells which line venous blood vessels, and are involved in functions that include, but are not limited to, angiogenesis, vascular permeability, vascular tone,</p>	<p>stimulating apoptosis of endothelial cells. An alternative highly preferred embodiment of the invention includes a method for inhibiting (e.g., decreasing) apoptosis of endothelial cells. A highly preferred embodiment of the invention includes a method for stimulating (e.g., increasing) endothelial cell activation. An alternative highly preferred embodiment of the invention includes a method for inhibiting (e.g., decreasing) the activation of and/or inactivating endothelial cells. A highly preferred embodiment of the invention includes a method for stimulating angiogenesis. An alternative highly preferred embodiment of the invention includes a method for inhibiting angiogenesis. A highly preferred embodiment of the invention includes a method for reducing cardiac hypertrophy. An alternative</p>
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					<p>highly preferred embodiment of the invention includes a method for inducing cardiac hypertrophy. Highly preferred indications include neoplastic diseases (e.g., as described below under “Hyperproliferative Disorders”), and disorders of the cardiovascular system (e.g., heart disease, congestive heart failure, hypertension, aortic stenosis, cardiomyopathy, valvular regurgitation, left ventricular dysfunction, atherosclerosis and atherosclerotic vascular disease, diabetic nephropathy, intracardiac shunt, cardiac hypertrophy, myocardial infarction, chronic hemodynamic overload, and/or as described below under “Cardiovascular Disorders”). Highly preferred indications include cardiovascular, endothelial and/or angiogenic disorders (e.g., systemic disorders that affect vessels such as diabetes mellitus, as</p>
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					<p>well as diseases of the vessels themselves, such as of the arteries, capillaries, veins and/or lymphatics). Highly preferred are indications that stimulate angiogenesis and/or cardiovascularization. Highly preferred are indications that inhibit angiogenesis and/or cardiovascularization.</p> <p>Highly preferred indications include antiangiogenic activity to treat solid tumors, leukemias, and Kaposi's sarcoma, and retinal disorders.</p> <p>Highly preferred indications include neoplasms and cancer, such as, Kaposi's sarcoma, hemangioma (capillary and cavernous), glomus tumors, telangiectasia, bacillary angiomatosis, hemangioendothelioma, angiosarcoma, haemangiopericytoma, lymphangioma, lymphangiosarcoma. Highly preferred indications also include cancers such as, prostate, breast, lung, colon,</p>
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					<p>pancreatic, esophageal, stomach, brain, liver, and urinary cancer. Preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Highly preferred indications also include arterial disease, such as, atherosclerosis, hypertension, coronary artery disease, inflammatory vasculitides, Reynaud's disease and Reynaud's phenomenon, aneurysms, restenosis; venous and lymphatic disorders such as thrombophlebitis, lymphangitis, and lymphedema; and other vascular disorders such as peripheral vascular disease, and cancer. Highly preferred indications also include trauma such as wounds, burns, and injured tissue (e.g., vascular injury such as, injury resulting from balloon angioplasty, and</p>
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					<p>atherosclerotic lesions), implant fixation, scarring, ischemia reperfusion injury, rheumatoid arthritis, cerebrovascular disease, renal diseases such as acute renal failure, and osteoporosis. Additional highly preferred indications include stroke, graft rejection, diabetic or other retinopathies, thrombotic and coagulative disorders, vasculitis, lymph angiogenesis, sexual disorders, age-related macular degeneration, and treatment /prevention of endometriosis and related conditions. Additional highly preferred indications include fibromas, heart disease, cardiac arrest, heart valve disease, and vascular disease. Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Preferred indications include</p>
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44	HDQHD03	348	Production of IL-10 and activation of T-cells.	Assays for production of IL-10 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of	autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Additional preferred indications include inflammation and inflammatory disorders (such as acute and chronic inflammatory diseases, e.g., inflammatory bowel disease and Crohn's disease), and pain management.
				Highly preferred indications include allergy and asthma. Additional highly preferred indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T	

				<p>the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and</p>	<p>cell-mediated immune response, and suppressing a T cell-mediated immune response.</p>
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45	HE2EN04	349	Activation of JNK Signaling Pathway in immune cells (such as eosinophils).	pathogenesis of allergy and asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.	Highly preferred indications include asthma, allergy, hypersensitivity reactions, inflammation, and inflammatory disorders. Additional highly preferred indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below). Highly preferred indications also include boosting or inhibiting immune cell proliferation. Preferred indications include neoplastic diseases (e.g.,
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				<p>1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. Exemplary cells that may be used according to these assays include eosinophils. Eosinophils are important in the late stage of allergic reactions; they are recruited to tissues and mediate the inflammatory response of late stage allergic reaction. Moreover, exemplary assays that may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate signal transduction, cell proliferation, activation, or apoptosis in eosinophils</p>	<p>leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include boosting an eosinophil-mediated immune response, and suppressing an eosinophil-mediated immune response.</p>
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				<p>include assays disclosed and/or cited in: Zhang JP, et al., "Role of caspases in dexamethasone-induced apoptosis and activation of c-Jun NH2-terminal kinase and p38 mitogen-activated protein kinase in human eosinophils" Clin Exp Immunol; Oct;122(1):20-7 (2000); Hebestreit H, et al., "Disruption of fas receptor signaling by nitric oxide in eosinophils" J Exp Med; Feb 2;187(3):415-25 (1998); J Allergy Clin Immunol 1999 Sep;104(3 Pt 1):565-74; and, Sousa AR, et al., "In vivo resistance to corticosteroids in bronchial asthma is associated with enhanced phosphorylation of JUN N-terminal kinase and failure of prednisolone to inhibit JUN N-terminal kinase phosphorylation" J Allergy Clin Immunol; Sep;104(3 Pt 1):565-74 (1999); the contents of each of which are herein incorporated by reference in its</p>
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46	HE8QV67	350	Production of IL-4	entirety. IL-4 FMAT. Assays for immunomodulatory proteins secreted by TH2 cells that stimulate B cells, T cells, macrophages and mast cells and promote polarization of CD4+ cells into TH2 cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation, stimulate immune cells, modulate immune cell polarization, and/or mediate humoral or cell-mediated immunity. Exemplary assays that test for immunomodulatory proteins evaluate the production of cytokines, such as IL-4, and the stimulation of immune cells, such as B cells, T cells, macrophages and mast cells. Such assays that may be used or routinely modified to test immunomodulatory activity of	A highly preferred embodiment of the invention includes a method for stimulating (e.g., increasing) IL-4 production. An alternative highly preferred embodiment of the invention includes a method for inhibiting (e.g., reducing) IL-4 production. A highly preferred indication includes asthma. A highly preferred indication includes allergy. A highly preferred indication includes rhinitis. Additional highly preferred indications include inflammation and inflammatory disorders. Highly preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, melanoma, and/or as described below under "Hyperproliferative Disorders"). Preferred indications include neoplasms and cancers, such as, for example, leukemia, lymphoma, melanoma, and prostate,
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				<p>polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204 (1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); Gonzalez et al., J Clin Lab Anal 8(5):277-283 (1994); Yssel et al., Res Immunol 144(8):610-616 (1993); Bagley et al., Nat Immunol 1(3):257-261 (2000); and van der Graaff et al., Rheumatology (Oxford) 38(3):214-220 (1999), the contents of each of which are herein incorporated by reference in its entirety. Human T cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human T cells are primary human lymphocytes that mature in the thymus and express a T cell receptor and CD3, CD4, or CD8. These</p>	<p>breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma,</p>
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				cells mediate humoral or cell-mediated immunity and may be preactivated to enhance responsiveness to immunomodulatory factors.	arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, and Lyme Disease. An additional preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease").
47	HE8UB86	351	Production of IL-10 and activation of T-cells.	Assays for production of IL-10 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of	Highly preferred indications include allergy and asthma. Additional highly preferred indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T

				<p>the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and</p>	<p>cell-mediated immune response, and suppressing a T cell-mediated immune response.</p>
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48	HE9BK23	352	<p>Activation of transcription through NFKB response element in immune cells (such as T-cells).</p>	<p>pathogenesis of allergy and asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.</p>	<p>Assays for the activation of transcription through the NFKB response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription through the NFKB response element that may be used or routinely modified to test NFKB-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays</p>	<p>Highly preferred indications include inflammation and inflammatory disorders. Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), and immunodeficiencies (e.g., as described below). An additional highly preferred indication is infection (e.g., AIDS, and/or an infectious disease as described below under "Infectious Disease"). Highly preferred indications</p>
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				<p>disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Black et al., Virus Gnes 15(2):105-117 (1997); and Fraser et al., 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. Exemplary human T cells, such as the MOLT4, that may be used according to these assays are publicly available (e.g., through the ATCC).</p>	<p>include neoplastic diseases (e.g., melanoma, leukemia, lymphoma, and/or as described below under “Hyperproliferative Disorders”). Highly preferred indications include neoplasms and cancers, such as, for example, melanoma, renal cell carcinoma, leukemia, lymphoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications also include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin’s disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt’s lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia,</p>
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					neutrophilia, psoriasis, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, suppression of immune reactions to transplanted organs, asthma and allergy.
48	HE9BK23	352	Activation of transcription through CD28 response element in immune cells (such as T-cells).	Assays for the activation of transcription through the CD28 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate IL-2 expression in T cells. Exemplary assays for transcription through the CD28 response element that may be used or routinely modified to test CD28-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol	<p>A highly preferred embodiment of the invention includes a method for stimulating T cell proliferation. An alternative highly preferred embodiment of the invention includes a method for inhibiting T cell proliferation. A highly preferred embodiment of the invention includes a method for activating T cells. An alternative highly preferred embodiment of the invention includes a method for inhibiting the activation of and/or inactivating T cells. A highly preferred embodiment of the invention includes a method for stimulating (e.g., increasing) IL-2 production. An alternative highly preferred embodiment</p>

				<p>216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); McGuire and Iacobelli, J Immunol 159(3):1319-1327 (1997); Parra et al., J Immunol 166(4):2437-2443 (2001); and Butscher et al., J Biol Chem 3(1):552-560 (1998), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.</p>	<p>of the invention includes a method for inhibiting (e.g., reducing) IL-2 production. Additional highly preferred indications include inflammation and inflammatory disorders. Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Highly preferred indications include neoplastic diseases (e.g., melanoma, renal cell carcinoma, leukemia, lymphoma, and/or as described below under “Hyperproliferative Disorders”). Highly preferred indications include neoplasms and cancers, such as, for example, melanoma (e.g., metastatic melanoma), renal</p>
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					<p>cell carcinoma (e.g., metastatic renal cell carcinoma), leukemia, lymphoma (e.g., T cell lymphoma), and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. A highly preferred indication includes infection (e.g., AIDS, tuberculosis, infections associated with granulomatous disease, and osteoporosis, and/or as described below under "Infectious Disease"). A highly preferred indication is AIDS. Additional highly preferred indications include suppression of immune reactions to transplanted organs and/or tissues, uveitis, psoriasis, and tropical spastic paraparesis. Preferred indications include blood disorders (e.g., as described</p>
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					below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Preferred indications also include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, asthma and allergy.
49	HEBBN36	353	Regulation of apoptosis of immune cells (such as mast cells).	Caspase Apoptosis. Assays for caspase apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate caspase protease-mediated apoptosis in immune cells (such as, for	Preferred embodiments of the invention include using polypeptides of the invention (or antibodies, agonists, or antagonists thereof) in detection, diagnosis, prevention, and/or treatment of asthma, allergy, hypersensitivity and inflammation.

				<p>example, in mast cells). Mast cells are found in connective and mucosal tissues throughout the body, and their activation via immunoglobulin E - antigen, promoted by T helper cell type 2 cytokines, is an important component of allergic disease. Dysregulation of mast cell apoptosis may play a role in allergic disease and mast cell tumor survival. Exemplary assays for caspase apoptosis that may be used or routinely modified to test caspase apoptosis activity induced by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in: Masuda A, et al., J Biol Chem, 276(28):26107-26113 (2001); Yeatman CF 2nd, et al., J Exp Med, 192(8):1093-1103 (2000); Lee et al., FEBS Lett 485(2-3): 122-126 (2000); Nor et al., J Vasc Res 37(3): 209-218 (2000); and Karsan and Harlan, J Atheroscler Thromb</p>
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50	HEQCC55	354	Production of MCP-1	<p>3(2): 75-80 (1996); the contents of each of which are herein incorporated by reference in its entirety. Immune cells that may be used according to these assays are publicly available (e.g., through commercial sources). Exemplary immune cells that may be used according to these assays include mast cells such as the HMC human mast cell line.</p> <p>MCP-1 FMAT. Assays for immunomodulatory proteins that are produced by a large variety of cells and act to induce chemotaxis and activation of monocytes and T cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation, induce chemotaxis, and modulate immune cell activation. Exemplary assays that test for</p>	<p>A highly preferred embodiment of the invention includes a method for stimulating (e.g., increasing) MCP-1 production. An alternative highly preferred embodiment of the invention includes a method for inhibiting (e.g., reducing) MCP-1 production. A highly preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease"). Additional highly preferred indications include inflammation and</p>
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				<p>immunomodulatory proteins evaluate the production of cell surface markers, such as monocyte chemoattractant protein (MCP), and the activation of monocytes and T cells. Such assays that may be used or routinely modified to test immunomodulatory and differentiation activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204(1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); Sathaporn and Eremis, J R Coll Surg Ednb 45(1):9-19 (2001); and Verhasselt et al., J Immunol 158:2919-2925 (1997), the contents of each of which are herein incorporated by reference in its entirety. Human dendritic cells that may be used according to these assays may be isolated using</p>	<p>inflammatory disorders. Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Preferred indications also include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation,</p>
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				<p>techniques disclosed herein or otherwise known in the art. Human dendritic cells are antigen presenting cells in suspension culture, which, when activated by antigen and/or cytokines, initiate and upregulate T cell proliferation and functional activities.</p>	<p>diabetes mellitus, endocarditis, meningitis (bacterial and viral), Lyme Disease, asthma, and allergy Preferred indications also include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, leukemia, lymphoma, prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver, and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia.</p>
50	HEQCC55	354	<p>Production of IL-13 and activation of T-cells.</p>	<p>Assays for production of IL-13 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the</p>	<p>Highly preferred indications include allergy and asthma. Additional highly preferred indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"),</p>

				<p>invention) to stimulate or inhibit production of IL-13 and/or activation of T-cells. Exemplary assays for IL-13 production that may be used or routinely modified to test activity of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) include, for example, assays such as disclosed and/or cited in: Grunig, G, et al., "Requirement for IL-13 independently of IL-4 in Experimental asthma" Science;282: 2261-2263 (1998), and Wills-Karp M, et al., "Interleukin-13: central mediator of allergic asthma" Science; 282: 2258-2261 (1998); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL13, a Th2 type cytokine, is a potent stimulus for mucus production, airway hyper-responsiveness</p>	<p>autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response.</p>
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				and allergic asthma. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated in in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.	
51	HESAJ10	355	Regulation of apoptosis of immune cells (such as mast cells).	Caspase Apoptosis. Assays for caspase apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate caspase protease-mediated apoptosis in immune cells (such as, for example, in mast cells). Mast cells are found in connective and mucosal tissues throughout the body, and their activation via immunoglobulin E -	Preferred embodiments of the invention include using polypeptides of the invention (or antibodies, agonists, or antagonists thereof) in detection, diagnosis, prevention, and/or treatment of asthma, allergy, hypersensitivity and inflammation.

				<p>antigen, promoted by T helper cell type 2 cytokines, is an important component of allergic disease. Dysregulation of mast cell apoptosis may play a role in allergic disease and mast cell tumor survival. Exemplary assays for caspase apoptosis that may be used or routinely modified to test caspase apoptosis activity induced by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in: Masuda A, et al., J Biol Chem, 276(28):26107-26113 (2001); Yeatman CF 2nd, et al., J Exp Med, 192(8):1093-1103 (2000); Lee et al., FEBS Lett 485(2-3): 122-126 (2000); Nor et al., J Vasc Res 37(3): 209-218 (2000); and Karsan and Harlan, J Atheroscler Thromb 3(2): 75-80 (1996); the contents of each of which are herein incorporated by reference in its entirety. Immune cells that may be used</p>	
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				according to these assays are publicly available (e.g., through commercial sources). Exemplary immune cells that may be used according to these assays include mast cells such as the HMC human mast cell line.	
52	HETEU28	356	Production of IL-5	IL-5 FMAT. Assays for immunomodulatory proteins secreted by TH2 cells, mast cells, basophils, and eosinophils that stimulate eosinophil function and B cell Ig production and promote polarization of CD4+ cells into TH2 cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation, stimulate immune cell function, modulate B cell Ig production, modulate immune cell polarization, and/or mediate humoral or cell-mediated immunity.	A highly preferred embodiment of the invention includes a method for inhibiting (e.g., reducing) IL-5 production. An alternative highly preferred embodiment of the invention includes a method for stimulating (e.g., increasing) IL-5 production. A highly preferred embodiment of the invention includes a method for stimulating (e.g., increasing) immunoglobulin production. An alternative highly preferred embodiment of the invention includes a method for inhibiting (e.g., decreasing) immunoglobulin production. A highly preferred indication includes allergy. A highly preferred indication includes

				<p>Exemplary assays that test for immunomodulatory proteins evaluate the production of cytokines, such as IL-5, and the stimulation of eosinophil function and B cell Ig production. Such assays that may be used or routinely modified to test immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204 (1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); Ohshima et al., Blood 92(9):3338-3345 (1998); Jung et al., Eur J Immunol 25(8):2413-2416 (1995); Mori et al., J Allergy Clin Immunol 106(1 Pt 2):558-564 (2000); and Koning et al., Cytokine 9(6):427-436 (1997), the contents of each of which are herein incorporated by reference in its entirety.</p>	<p>asthma. A highly preferred indication includes rhinitis. An additional highly preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease"), and inflammation and inflammatory disorders. Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, melanoma, and/or as described below under "Hyperproliferative Disorders"). Preferred indications include neoplasms and cancers, such as, leukemia,</p>
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				<p>Human T cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human T cells are primary human lymphocytes that mature in the thymus and express a T cell receptor and CD3, CD4, or CD8. These cells mediate humoral or cell-mediated immunity and may be preactivated to enhance responsiveness to immunomodulatory factors.</p>	<p>lymphoma, melanoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, leukemias, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, and Lyme Disease.</p>
52	HETEU28	356	SEAP in OE-33		

53	HFABG18	357	Activation of Adipocyte ERK Signaling Pathway	<p>Kinase assay. Kinase assays, for example an Elk-1 kinase assay, for ERK signal transduction that regulate cell proliferation or differentiation are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and differentiation. Exemplary assays for ERK kinase activity that may be used or routinely modified to test ERK kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Le Marchand-Brustel Y, Exp Clin Endocrinol Diabetes 107(2):126-132 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang</p>	<p>A highly preferred embodiment of the invention includes a method for stimulating adipocyte proliferation. An alternative highly preferred embodiment of the invention includes a method for inhibiting adipocyte proliferation. A highly preferred embodiment of the invention includes a method for stimulating adipocyte differentiation. An alternative highly preferred embodiment of the invention includes a method for inhibiting adipocyte differentiation. A highly preferred embodiment of the invention includes a method for stimulating (e.g., increasing) adipocyte activation. An alternative highly preferred embodiment of the invention includes a method for inhibiting the activation of (e.g., decreasing) and/or inactivating adipocytes. Highly preferred indications include endocrine disorders</p>
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				<p>and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety.</p> <p>Mouse adipocyte cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary mouse adipocyte cells that may be used according to these assays include 3T3-L1 cells. 3T3-L1 is an adherent mouse preadipocyte cell line that is a continuous substrain of 3T3 fibroblast cells developed through clonal isolation and undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation conditions known in the art.</p>	<p>(e.g., as described below under "Endocrine Disorders").</p> <p>Highly preferred indications also include neoplastic diseases (e.g., lipomas, liposarcomas, and/or as described below under "Hyperproliferative Disorders"). Preferred indications include blood disorders (e.g., hypertension, congestive heart failure, blood vessel blockage, heart disease, stroke, impotence and/or as described below under "Immune Activity", "Cardiovascular Disorders", and/or "Blood-Related Disorders"), immune disorders (e.g., as described below under "Immune Activity"), neural disorders (e.g., as described below under "Neural Activity and Neurological Diseases"), and infection (e.g., as described below under "Infectious Disease").</p> <p>A highly preferred indication is diabetes mellitus. An additional highly preferred</p>
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					<p>indication is a complication associated with diabetes (e.g., diabetic retinopathy, diabetic nephropathy, kidney disease (e.g., renal failure, nephropathy and/or other diseases and disorders as described in the "Renal Disorders" section below), diabetic neuropathy, nerve disease and nerve damage (e.g., due to diabetic neuropathy), blood vessel blockage, heart disease, stroke, impotence (e.g., due to diabetic neuropathy or blood vessel blockage), seizures, mental confusion, drowsiness, nonketotic hyperglycemic-hyperosmolar coma, cardiovascular disease (e.g., heart disease, atherosclerosis, microvascular disease, hypertension, stroke, and other diseases and disorders as described in the "Cardiovascular Disorders" section below), dyslipidemia, endocrine disorders (as described in the "Endocrine</p>
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				Disorders" section below), neuropathy, vision impairment (e.g., diabetic retinopathy and blindness), ulcers and impaired wound healing, infection (e.g., infectious diseases and disorders as described in the "Infectious Diseases" section below (particularly of the urinary tract and skin). An additional highly preferred indication is obesity and/or complications associated with obesity. Additional highly preferred indications include weight loss or alternatively, weight gain. Additional highly preferred indications are complications associated with insulin resistance. Additional highly preferred indications are disorders of the musculoskeletal systems including myopathies, muscular dystrophy, and/or as described herein. Additional highly preferred indications include, hypertension, coronary artery disease, dyslipidemia,
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53	HFABG18	357	Protection from Endothelial Cell Apoptosis.	Caspase Apoptosis Rescue. Assays for caspase apoptosis rescue are well known in the art and may be used or routinely modified to assess the ability of the polypeptides of the invention (including antibodies and agonists or	gallstones, osteoarthritis, degenerative arthritis, eating disorders, fibrosis, cachexia, and kidney diseases or disorders. Preferred indications include neoplasms and cancer, such as, lymphoma, leukemia and breast, colon, and kidney cancer. Additional preferred indications include melanoma, prostate, lung, pancreatic, esophageal, stomach, brain, liver, and urinary cancer. Highly preferred indications include lipomas and liposarcomas. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia.
					A highly preferred embodiment of the invention includes a method for stimulating endothelial cell growth. An alternative highly preferred embodiment of the invention includes a method for inhibiting endothelial cell

			<p>antagonists of the invention) to inhibit caspase protease-mediated apoptosis. Exemplary assays for caspase apoptosis that may be used or routinely modified to test caspase apoptosis rescue of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Romeo et al., Cardiovasc Res 45(3): 788-794 (2000); Messmer et al., Br J Pharmacol 127(7): 1633-1640 (1999); and J Atheroscler Thromb 3(2): 75-80 (1996); the contents of each of which are herein incorporated by reference in its entirety. Endothelial cells that may be used according to these assays are publicly available (e.g., through commercial sources). Exemplary endothelial cells that may be used according to these assays include bovine aortic endothelial cells (bAEC), which are an example of endothelial cells which line</p>	<p>growth. A highly preferred embodiment of the invention includes a method for stimulating endothelial cell proliferation. An alternative highly preferred embodiment of the invention includes a method for inhibiting endothelial cell proliferation. A highly preferred embodiment of the invention includes a method for stimulating endothelial cell growth. An alternative highly preferred embodiment of the invention includes a method for inhibiting endothelial cell growth. A highly preferred embodiment of the invention includes a method for stimulating apoptosis of endothelial cells. An alternative highly preferred embodiment of the invention includes a method for inhibiting (e.g., decreasing) apoptosis of endothelial cells. A highly preferred embodiment of the invention includes a method for</p>
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				<p>blood vessels and are involved in functions that include, but are not limited to, angiogenesis, vascular permeability, vascular tone, and immune cell extravasation.</p>	<p>stimulating angiogenesis. An alternative highly preferred embodiment of the invention includes a method for inhibiting angiogenesis. A highly preferred embodiment of the invention includes a method for reducing cardiac hypertrophy. An alternative highly preferred embodiment of the invention includes a method for inducing cardiac hypertrophy. Highly preferred indications include neoplastic diseases (e.g., as described below under "Hyperproliferative Disorders"), and disorders of the cardiovascular system (e.g., heart disease, congestive heart failure, hypertension, aortic stenosis, cardiomyopathy, valvular regurgitation, left ventricular dysfunction, atherosclerosis and atherosclerotic vascular disease, diabetic nephropathy, intracardiac shunt, cardiac hypertrophy, myocardial infarction, chronic</p>
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					hemodynamic overload, and/or as described below under “Cardiovascular Disorders”). Highly preferred indications include cardiovascular, endothelial and/or angiogenic disorders (e.g., systemic disorders that affect vessels such as diabetes mellitus, as well as diseases of the vessels themselves, such as of the arteries, capillaries, veins and/or lymphatics). Highly preferred are indications that stimulate angiogenesis and/or cardiovascularization. Highly preferred are indications that inhibit angiogenesis and/or cardiovascularization. Highly preferred indications include antiangiogenic activity to treat solid tumors, leukemias, and Kaposi's sarcoma, and retinal disorders. Highly preferred indications include neoplasms and cancer, such as, Kaposi's sarcoma, hemangioma (capillary and cavernous), glomus tumors, telangiectasia, bacillary
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					<p>angiomas, hemangiomas, angiosarcoma, haemangiopericytoma, lymphangioma, lymphangiosarcoma. Highly preferred indications also include cancers such as, prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver, and urinary cancer. Preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Highly preferred indications also include arterial disease, such as, atherosclerosis, hypertension, coronary artery disease, inflammatory vasculitides, Reynaud's disease and Reynaud's phenomenon, aneurysms, restenosis; venous and lymphatic disorders such as thrombophlebitis, lymphangitis, and lymphedema; and other</p>
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					<p>vascular disorders such as peripheral vascular disease, and cancer. Highly preferred indications also include trauma such as wounds, burns, and injured tissue (e.g., vascular injury such as, injury resulting from balloon angioplasty, and atherosclerotic lesions), implant fixation, scarring, ischemia reperfusion injury, rheumatoid arthritis, cerebrovascular disease, renal diseases such as acute renal failure, and osteoporosis. Additional highly preferred indications include stroke, graft rejection, diabetic or other retinopathies, thrombotic and coagulative disorders, vasculitis, lymph angiogenesis, sexual disorders, age-related macular degeneration, and treatment /prevention of endometriosis and related conditions. Additional highly preferred indications include fibromas, heart disease, cardiac arrest,</p>
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					heart valve disease, and vascular disease. Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Additional preferred indications include inflammation and inflammatory disorders (such as acute and chronic inflammatory diseases, e.g., inflammatory bowel disease and Crohn's disease), and pain management.
53	HFABG18	357	Production of IFNgamma using a T cells	IFNgamma FMAT. IFNγ plays a central role in the immune system and is considered to be a proinflammatory cytokine. IFNγ promotes TH1 and inhibits TH2 differentiation;	A highly preferred embodiment of the invention includes a method for stimulating the production of IFNγ. An alternative highly preferred embodiment of the

				<p>promotes IgG2a and inhibits IgE secretion; induces macrophage activation; and increases MHC expression. Assays for immunomodulatory proteins produced by T cells and NK cells that regulate a variety of inflammatory activities and inhibit TH2 helper cell functions are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation, regulate inflammatory activities, modulate TH2 helper cell function, and/or mediate humoral or cell-mediated immunity. Exemplary assays that test for immunomodulatory proteins evaluate the production of cytokines, such as Interferon gamma (IFNg), and the activation of T cells. Such assays that may be used or</p>	<p>invention includes a method for inhibiting the production of IFNg. Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"), and infection (e.g., viral infections, tuberculosis, infections associated with chronic granulomatous disease and malignant osteoporosis, and/or as described below under "Infectious Disease"). Highly preferred indications include autoimmune disease (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), immunodeficiency (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional highly preferred indications include inflammation and</p>
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			<p>routinely modified to test immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204 (1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); Gonzalez et al., J Clin Lab Anal 8(5):225-233 (1995); Billiau et al., Ann NY Acad Sci 856:22-32 (1998); Boehm et al., Annu Rev Immunol 15:749-795 (1997), and Rheumatology (Oxford) 38(3):214-20 (1999), the contents of each of which are herein incorporated by reference in its entirety. Human T cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human T cells are primary human lymphocytes that mature in the thymus and</p>	<p>inflammatory disorders. Additional preferred indications include idiopathic pulmonary fibrosis. Highly preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, melanoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, for example, leukemia, lymphoma, melanoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple</p>
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				express a T Cell receptor and CD3, CD4, or CD8. These cells mediate humoral or cell-mediated immunity and may be preactivated to enhance responsiveness to immunomodulatory factors.	myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, asthma and allergy.
54	HFAMB72	358	Activation of JNK Signaling Pathway in immune cells (such as eosinophils).	Kinase assay. JNK kinase assays for signal transduction that regulate cell proliferation, activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and apoptosis. Exemplary assays for JNK kinase activity that may be used or routinely modified to test JNK kinase-induced activity of polypeptides of the	Highly preferred indications include asthma, allergy, hypersensitivity reactions, inflammation, and inflammatory disorders. Additional highly preferred indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below). Highly

				<p>invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. Exemplary cells that may be used according to these assays include eosinophils. Eosinophils are important in the late stage of allergic reactions; they are recruited to tissues and mediate the inflammatory response of late stage allergic reaction. Moreover, exemplary assays that may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies</p>	<p>preferred indications also include boosting or inhibiting immune cell proliferation. Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include boosting an eosinophil-mediated immune response, and suppressing an eosinophil-mediated immune response.</p>
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				<p>and agonists or antagonists of the invention) to modulate signal transduction, cell proliferation, activation, or apoptosis in eosinophils include assays disclosed and/or cited in: Zhang JP, et al., "Role of caspases in dexamethasone-induced apoptosis and activation of c-Jun NH2-terminal kinase and p38 mitogen-activated protein kinase in human eosinophils" Clin Exp Immunol; Oct;122(1):20-7 (2000); Hebestreit H, et al., "Disruption of fas receptor signaling by nitric oxide in eosinophils" J Exp Med; Feb 2;187(3):415-25 (1998); J Allergy Clin Immunol 1999 Sep;104(3 Pt 1):565-74; and, Sousa AR, et al., "In vivo resistance to corticosteroids in bronchial asthma is associated with enhanced phosphorylation of JUN N-terminal kinase and failure of prednisolone to inhibit JUN N-terminal kinase</p>				
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55	HFCCQ50	359	Production of TNF alpha by dendritic cells	phosphorylation" J Allergy Clin Immunol; Sep;104(3 Pt 1):565-74 (1999); the contents of each of which are herein incorporated by reference in its entirety.	<p>A highly preferred embodiment of the invention includes a method for inhibiting (e.g., decreasing) TNF alpha production. An alternative highly preferred embodiment of the invention includes a method for stimulating (e.g., increasing) TNF alpha production. Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below),</p> <p>TNFa FMAT. Assays for immunomodulatory proteins produced by activated macrophages, T cells, fibroblasts, smooth muscle, and other cell types that exert a wide variety of inflammatory and cytotoxic effects on a variety of cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation, modulate inflammation and cytotoxicity. Exemplary assays that test for immunomodulatory proteins evaluate the production of cytokines such as tumor necrosis factor alpha (TNFa), and the induction or inhibition</p>
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				<p>of an inflammatory or cytotoxic response. Such assays that may be used or routinely modified to test immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204(1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); Verhasselt et al., Eur J Immunol 28(11):3886-3890 (1998); Dahlen et al., J Immunol 160(7):3585-3593 (1998); Verhasselt et al., J Immunol 158:2919-2925 (1997); and Nardelli et al., J Leukoc Biol 65:822-828 (1999), the contents of each of which are herein incorporated by reference in its entirety. Human dendritic cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art.</p>	<p>boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional highly preferred indications include inflammation and inflammatory disorders, and treating joint damage in patients with rheumatoid arthritis. An additional highly preferred indication is sepsis. Highly preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Additionally, highly preferred indications include neoplasms and cancers, such as, leukemia, lymphoma, melanoma, glioma (e.g., malignant glioma), solid tumors, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for</p>
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				Human dendritic cells are antigen presenting cells in suspension culture, which, when activated by antigen and/or cytokines, initiate and upregulate T cell proliferation and functional activities.	example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, cardiac reperfusion injury, and asthma and allergy. An additional preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease").
55	HFCCQ50	359	Production of IL-4	IL-4 FMAT. Assays for immunomodulatory proteins secreted by TH2 cells that stimulate B cells, T cells, macrophages and mast cells	A highly preferred embodiment of the invention includes a method for stimulating (e.g., increasing) IL-4 production. An alternative

			<p>and promote polarization of CD4+ cells into TH2 cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation, stimulate immune cells, modulate immune cell polarization, and/or mediate humoral or cell-mediated immunity. Exemplary assays that test for immunomodulatory proteins evaluate the production of cytokines, such as IL-4, and the stimulation of immune cells, such as B cells, T cells, macrophages and mast cells. Such assays that may be used or routinely modified to test immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-</p>	<p>highly preferred embodiment of the invention includes a method for inhibiting (e.g., reducing) IL-4 production. A highly preferred indication includes asthma. A highly preferred indication includes allergy. A highly preferred indication includes rhinitis. Additional highly preferred indications include inflammation and inflammatory disorders. Highly preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, melanoma, and/or as described below under "Hyperproliferative Disorders"). Preferred indications include neoplasms and cancers, such as, for example, leukemia, lymphoma, melanoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic</p>
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			<p>204 (1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); Gonzalez et al., J Clin Lab Anal 8(5):277-283 (1994); Yssel et al., Res Immunol 144(8):610-616 (1993); Bagley et al., Nat Immunol 1(3):257-261 (2000); and van der Graaff et al., Rheumatology (Oxford) 38(3):214-220 (1999), the contents of each of which are herein incorporated by reference in its entirety.</p> <p>Human T cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art.</p> <p>Human T cells are primary human lymphocytes that mature in the thymus and express a T cell receptor and CD3, CD4, or CD8. These cells mediate humoral or cell-mediated immunity and may be preactivated to enhance responsiveness to immunomodulatory factors.</p>	<p>conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted</p>
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55	HFCCQ50	359	Activation of transcription through NFKB response element in immune cells (such as the Jurkat human T cell line).	Assays for the activation of transcription through the NFKB response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription through the NFKB response element that may be used or routinely modified to test NFKB-response element activity of polypeptides of the invention (including antibodies and	organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, and Lyme Disease. An additional preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease").
				Highly preferred indications include inflammation and inflammatory disorders. Highly preferred indications include immunological and hematopoietic disorders (e.g., as described below under "Immune Activity"; "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), and immunodeficiencies (e.g., as described below). An additional highly preferred indication is infection (e.g., AIDS, and/or an infectious	

			<p>agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Valle Blazquez et al, Immunology 90(3):455-460 (1997); Aramburau et al., J Exp Med 82(3):801-810 (1995); and Fraser et al., 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the JURKAT cell line, which is a suspension culture of leukemia cells that produce IL-2 when stimulated.</p>	<p>disease as described below under "Infectious Disease"). Highly preferred indications include neoplastic diseases (e.g., melanoma, leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, melanoma, renal cell carcinoma, leukemia, lymphoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications also include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma,</p>
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					<p>arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, suppression of immune reactions to transplanted organs, asthma and allergy.</p>
55	HFCCQ50	359	<p>Activation of transcription through GAS response element in immune cells (such as monocytes).</p>	<p>Assays for the activation of transcription through the Gamma Interferon Activation Site (GAS) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate STAT transcription factors and modulate gene expression involved in a wide variety of cell functions. Exemplary assays for transcription through the GAS response element that may be used or routinely modified to test</p>	<p>Preferred embodiments of the invention include using polypeptides of the invention (or antibodies, agonists, or antagonists thereof) in detection, diagnosis, prevention, and/or treatment of Inflammation, Infection, Cancer, Hypersensitivity, and Atherosclerosis.</p>

				<p>GAS-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Gustafson KS, et al., J Biol Chem, 271(33):20035-20046 (1996); Eilers A, et al., Immunobiology, 193(2-4):328-333 (1995); Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Matikainen et al., Blood 93(6):1980-1991 (1999); and Henttinen et al., J Immunol 155(10):4582-4587 (1995), the contents of each of which are herein incorporated by reference in its entirety. Exemplary immune cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary immune cells that may be used according to these assays include the U937 cell</p>	
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56	HFIUZ70	360	Activation of JNK Signaling Pathway in immune cells (such as eosinophils).	line, which is a monocytic cell line.	Highly preferred indications include asthma, allergy, hypersensitivity reactions, inflammation, and inflammatory disorders. Additional highly preferred indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below). Highly preferred indications also include boosting or inhibiting immune cell proliferation. Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include boosting an
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				<p>410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. Exemplary cells that may be used according to these assays include eosinophils. Eosinophils are important in the late stage of allergic reactions; they are recruited to tissues and mediate the inflammatory response of late stage allergic reaction. Moreover, exemplary assays that may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate signal transduction, cell proliferation, activation, or apoptosis in eosinophils include assays disclosed and/or cited in: Zhang JP, et al., "Role of caspases in dexamethasone-induced apoptosis and activation of c-Jun NH2-</p>	eosinophil-mediated immune response, and suppressing an eosinophil-mediated immune response.
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				terminal kinase and p38 mitogen-activated protein kinase in human eosinophils" Clin Exp Immunol; Oct;122(1):20-7 (2000); Hebestreit H, et al., "Disruption of fas receptor signaling by nitric oxide in eosinophils" J Exp Med; Feb 2;187(3):415-25 (1998); J Allergy Clin Immunol 1999 Sep;104(3 Pt 1):565-74; and, Sousa AR, et al., "In vivo resistance to corticosteroids in bronchial asthma is associated with enhanced phosphorylation of JUN N-terminal kinase and failure of prednisolone to inhibit JUN N-terminal kinase phosphorylation" J Allergy Clin Immunol; Sep;104(3 Pt 1):565-74 (1999); the contents of each of which are herein incorporated by reference in its entirety.	
57	HFKET18	361	Regulation of apoptosis of immune cells (such as mast cells).	Caspase Apoptosis. Assays for caspase apoptosis are well known in the art and may be used or routinely modified to	Preferred embodiments of the invention include using polypeptides of the invention (or antibodies, agonists, or

				<p>assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate caspase protease-mediated apoptosis in immune cells (such as, for example, in mast cells). Mast cells are found in connective and mucosal tissues throughout the body, and their activation via immunoglobulin E - antigen, promoted by T helper cell type 2 cytokines, is an important component of allergic disease. Dysregulation of mast cell apoptosis may play a role in allergic disease and mast cell tumor survival. Exemplary assays for caspase apoptosis that may be used or routinely modified to test caspase apoptosis activity induced by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in: Masuda A, et al., J Biol Chem, 276(28):26107-26113 (2001);</p>	<p>antagonists thereof) in detection, diagnosis, prevention, and/or treatment of asthma, allergy, hypersensitivity and inflammation.</p>
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57	HFKET18	361	<p>Activation of transcription through NFAT response in immune cells (such as T-cells).</p>	<p>Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of</p>	<p>Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus,</p>

			<p>the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Serfling et al., Biochim Biophys Acta 1498(1):1-18 (2000); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Fraser et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J Biol Chem 268(19):14285-14293 (1993), the contents of each of which</p>	<p>multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional highly preferred indications include inflammation and inflammatory disorders. An additional highly preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease"). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Preferred indications include neoplasms and cancers, such as, for example, leukemia, lymphoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign</p>
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				are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the JURKAT cell line, which is a suspension culture of leukemia cells that produce IL-2 when stimulated.	dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications also include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, asthma and allergy.
57	HFKET18	361	Activation of Natural Killer Cell ERK Signaling Pathway.	Kinase assay. Kinase assays, for example an Elk-1 kinase assay, for ERK signal transduction that regulate cell proliferation or differentiation are well known in the art and may be used or routinely modified to assess the ability	A highly preferred embodiment of the invention includes a method for stimulating natural killer cell proliferation. An alternative highly preferred embodiment of the invention includes a method for inhibiting natural

				<p>of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and differentiation. Exemplary assays for ERK kinase activity that may be used or routinely modified to test ERK kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. Natural killer cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary natural</p>	<p>killer cell proliferation. A highly preferred embodiment of the invention includes a method for stimulating natural killer cell differentiation. An alternative highly preferred embodiment of the invention includes a method for inhibiting natural killer cell differentiation. Highly preferred indications include neoplastic diseases (e.g., as described below under "Hyperproliferative Disorders"), blood disorders (e.g., as described below under "Immune Activity", "Cardiovascular Disorders", and/or "Blood-Related Disorders"), immune disorders (e.g., as described below under "Immune Activity") and infections (e.g., as described below under "Infectious Disease"). Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or</p>
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				<p>killer cells that may be used according to these assays include the human natural killer cell lines (for example, NK-YT cells which have cytolytic and cytotoxic activity) or primary NK cells.</p>	<p>"Cardiovascular Disorders"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Additional highly preferred indications include inflammation and inflammatory disorders. Highly preferred indications also include cancers such as, kidney, melanoma, prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver, urinary cancer, lymphoma and leukemias. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Other highly preferred indications include, pancytopenia, leukopenia, leukemias, Hodgkin's disease, acute lymphocytic anemia</p>
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					(ALL), arthritis, asthma, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, psoriasis, immune reactions to transplanted organs and tissues, endocarditis, meningitis, Lyme Disease, and allergies.
58	HFLNB64	362	Production of IL-5	IL-5 FMAT. Assays for immunomodulatory proteins secreted by TH2 cells, mast cells, basophils, and eosinophils that stimulate eosinophil function and B cell Ig production and promote polarization of CD4+ cells into TH2 cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation, stimulate immune cell function, modulate B cell Ig production, modulate immune cell polarization, and/or mediate humoral or cell-mediated immunity.	<p>A highly preferred embodiment of the invention includes a method for inhibiting (e.g., reducing) IL-5 production. An alternative highly preferred embodiment of the invention includes a method for stimulating (e.g., increasing) IL-5 production. A highly preferred embodiment of the invention includes a method for stimulating (e.g., increasing) immunoglobulin production. An alternative highly preferred embodiment of the invention includes a method for inhibiting (e.g., decreasing) immunoglobulin production. A highly preferred indication includes allergy. A highly preferred indication includes</p>

			<p>Exemplary assays that test for immunomodulatory proteins evaluate the production of cytokines, such as IL-5, and the stimulation of eosinophil function and B cell Ig production. Such assays that may be used or routinely modified to test immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204 (1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); Ohshima et al., Blood 92(9):3338-3345 (1998); Jung et al., Eur J Immunol 25(8):2413-2416 (1995); Mori et al., J Allergy Clin Immunol 106(1 Pt 2):558-564 (2000); and Koning et al., Cytokine 9(6):427-436 (1997), the contents of each of which are herein incorporated by reference in its entirety.</p>	<p>asthma. A highly preferred indication includes rhinitis. An additional highly preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease"), and inflammation and inflammatory disorders. Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, melanoma, and/or as described below under "Hyperproliferative Disorders"). Preferred indications include neoplasms and cancers, such as, leukemia,</p>
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				<p>Human T cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human T cells are primary human lymphocytes that mature in the thymus and express a T cell receptor and CD3, CD4, or CD8. These cells mediate humoral or cell-mediated immunity and may be preactivated to enhance responsiveness to immunomodulatory factors.</p>	<p>lymphoma, melanoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, leukemias, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, and Lyme Disease.</p>
59	HFOXA73	363	Production of IL-10 and activation of T-	Assays for production of IL-10 and activation of T-cells are	Highly preferred indications include allergy and asthma.

			<p>cells.</p>	<p>well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by</p>	<p>Additional highly preferred indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response.</p>
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				reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.	
60	HFPAC12	364	Regulation of apoptosis of immune cells (such as mast cells).	Caspase Apoptosis. Assays for caspase apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate caspase protease-mediated apoptosis in	Preferred embodiments of the invention include using polypeptides of the invention (or antibodies, agonists, or antagonists thereof) in detection, diagnosis, prevention, and/or treatment of asthma, allergy, hypersensitivity and inflammation.

				<p>immune cells (such as, for example, in mast cells). Mast cells are found in connective and mucosal tissues throughout the body, and their activation via immunoglobulin E - antigen, promoted by T helper cell type 2 cytokines, is an important component of allergic disease. Dysregulation of mast cell apoptosis may play a role in allergic disease and mast cell tumor survival. Exemplary assays for caspase apoptosis that may be used or routinely modified to test caspase apoptosis activity induced by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in: Masuda A, et al., J Biol Chem, 276(28):26107-26113 (2001); Yeatman CF 2nd, et al., J Exp Med, 192(8):1093-1103 (2000); Lee et al., FEBS Lett 485(2-3): 122-126 (2000); Nor et al., J Vasc Res 37(3): 209-218 (2000); and Karsan and</p>	
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61	HFPAO71	365	Activation of JNK Signaling Pathway in immune cells (such as eosinophils).	<p>Harlan, J Atheroscler Thromb 3(2): 75-80 (1996); the contents of each of which are herein incorporated by reference in its entirety.</p> <p>Immune cells that may be used according to these assays are publicly available (e.g., through commercial sources). Exemplary immune cells that may be used according to these assays include mast cells such as the HMC human mast cell line.</p>	<p>Highly preferred indications include asthma, allergy, hypersensitivity reactions, inflammation, and inflammatory disorders.</p> <p>Additional highly preferred indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below),</p>
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				<p>test JNK kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. Exemplary cells that may be used according to these assays include eosinophils. Eosinophils are important in the late stage of allergic reactions; they are recruited to tissues and mediate the inflammatory response of late stage allergic reaction. Moreover, exemplary assays that may be used or routinely modified to assess the ability</p>	<p>immunodeficiencies (e.g., as described below). Highly preferred indications also include boosting or inhibiting immune cell proliferation. Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include boosting an eosinophil-mediated immune response, and suppressing an eosinophil-mediated immune response.</p>
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				<p>of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate signal transduction, cell proliferation, activation, or apoptosis in eosinophils include assays disclosed and/or cited in: Zhang JP, et al., "Role of caspases in dexamethasone-induced apoptosis and activation of c-Jun NH2-terminal kinase and p38 mitogen-activated protein kinase in human eosinophils" Clin Exp Immunol; Oct;122(1):20-7 (2000); Hebestreit H, et al., "Disruption of fas receptor signaling by nitric oxide in eosinophils" J Exp Med; Feb 2;187(3):415-25 (1998); J Allergy Clin Immunol 1999 Sep;104(3 Pt 1):565-74; and, Sousa AR, et al., "In vivo resistance to corticosteroids in bronchial asthma is associated with enhanced phosphorylation of JUN N-terminal kinase and failure of</p>	
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61	HFPAO71	365	Production of IL-8 by immune cells (such as the human EOL-1 eosinophil cells)	prednisolone to inhibit JUN N-terminal kinase phosphorylation" J Allergy Clin Immunol; Sep;104(3 Pt 1):565-74 (1999); the contents of each of which are herein incorporated by reference in its entirety.	Highly preferred indications include eosinophilia, asthma, allergy, hypersensitivity reactions, inflammation, and inflammatory disorders. Additional highly preferred indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below). Highly preferred indications also include boosting or inhibiting immune cell proliferation. Preferred indications include
				Assay that measures the production of the chemokine interleukin-8 (IL-8) from immune cells (such as the EOL-1 human eosinophil cell line) are well known in the art (for example, measurement of IL-8 production by FMAT) and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit. Eosinophils are a type of immune cell important in allergic responses; they are recruited to tissues and mediate the inflammatory response of late stage allergic reaction. IL8 is a strong immunomodulator and may	

				have a potential proinflammatory role in immunological diseases and disorders (such as allergy and asthma).	neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include boosting an eosinophil-mediated immune response, and suppressing an eosinophil-mediated immune response.
61	HFPAO71	365	Production of IL-8 by endothelial cells (such as Human Umbilical Cord Endothelial Cells).	Assays measuring production of IL-8 are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate production and/or secretion of IL-8. For example, FMAT may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate production and/or secretion of IL-8 from endothelial cells (such as human umbilical vein	Highly preferred indications include immunological and inflammatory disorders (e.g., such as allergy, asthma, leukemia, etc. and as described below under "Immune Activity", and "Blood-Related Disorders"). Highly preferred indications also include autoimmune disorders (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), neoplastic disorders (e.g., organ cancers such as lung, liver, colon cancer, and/or as described below under "Hyperproliferative Disorders"), and

				endothelial cells (HUVEC)). HUVECs are endothelial cells which line venous blood vessels, and are involved in functions that include, but are not limited to, angiogenesis, vascular permeability, vascular tone, and immune cell extravasation. Endothelial cells play a pivotal role in the initiation and perpetuation of inflammation and secretion of IL-8 may play an important role in recruitment and activation of immune cells such as neutrophils, macrophages, and lymphocytes.	cardiovascular disorders (e.g. such as described below under "Cardiovascular Disorders"). Preferred indications include thrombosis, bacteremia and sepsis syndrome and consequent complications (such as acute respiratory distress syndrome and systemic ischemia-reperfusion resulting from septic shock), restenosis and atherosclerosis.
62	HFPCX36	366	SEAP in Senescence Assay		
62	HFPCX36	366	Activation of transcription through NFkB response element in immune cells (such as T-cells).	Assays for the activation of transcription through the NFkB response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFkB	Highly preferred indications include inflammation and inflammatory disorders. Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Highly preferred indications

			transcription factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription through the NFkB response element that may be used or routinely modified to test NFkB-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Black et al., Virus Gnes 15(2):105-117 (1997); and Fraser et al., 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that	include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), and immunodeficiencies (e.g., as described below). An additional highly preferred indication is infection (e.g., AIDS, and/or an infectious disease as described below under "Infectious Disease"). Highly preferred indications include neoplastic diseases (e.g., melanoma, leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, melanoma, renal cell carcinoma, leukemia, lymphoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic
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				may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.	conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications also include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, suppression of immune reactions to transplanted organs, asthma and allergy.
63	HFPCX64	367	Production of IL-5	IL-5 FMAT. Assays for immunomodulatory proteins secreted by TH2 cells, mast cells, basophils, and eosinophils that stimulate eosinophil function and B cell Ig production and promote polarization of CD4+ cells into TH2 cells are well known in	A highly preferred embodiment of the invention includes a method for inhibiting (e.g., reducing) IL-5 production. An alternative highly preferred embodiment of the invention includes a method for stimulating (e.g., increasing) IL-5 production.

				<p>the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation, stimulate immune cell function, modulate B cell Ig production, modulate immune cell polarization, and/or mediate humoral or cell-mediated immunity.</p> <p>Exemplary assays that test for immunomodulatory proteins evaluate the production of cytokines, such as IL-5, and the stimulation of eosinophil function and B cell Ig production. Such assays that may be used or routinely modified to test immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204 (1999); Rowland et al.,</p>	<p>A highly preferred embodiment of the invention includes a method for stimulating (e.g., increasing) immunoglobulin production. An alternative highly preferred embodiment of the invention includes a method for inhibiting (e.g., decreasing) immunoglobulin production. A highly preferred indication includes allergy. A highly preferred indication includes asthma. A highly preferred indication includes rhinitis. An additional highly preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease"), and inflammation and inflammatory disorders. Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic</p>
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				<p>"Lymphocytes: a practical approach" Chapter 6:138-160 (2000); Ohshima et al., Blood 92(9):3338-3345 (1998); Jung et al., Eur J Immunol 25(8):2413-2416 (1995); Mori et al., J Allergy Clin Immunol 106(1 Pt 2):558-564 (2000); and Koning et al., Cytokine 9(6):427-436 (1997), the contents of each of which are herein incorporated by reference in its entirety. Human T cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human T cells are primary human lymphocytes that mature in the thymus and express a T cell receptor and CD3, CD4, or CD8. These cells mediate humoral or cell-mediated immunity and may be preactivated to enhance responsiveness to immunomodulatory factors.</p>	<p>lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, melanoma, and/or as described below under "Hyperproliferative Disorders"). Preferred indications include neoplasms and cancers, such as, leukemia, lymphoma, melanoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, leukemias, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's</p>
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					lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, and Lyme Disease.
64	HFTBM50	368	Insulin Secretion	Assays for measuring secretion of insulin are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate insulin secretion. For example, insulin secretion is measured by FMAT using anti-rat insulin antibodies. Insulin secretion from pancreatic beta cells is upregulated by glucose and also by certain proteins/peptides, and dysregulation is a key component in diabetes.	A highly preferred indication is diabetes mellitus. An additional highly preferred indication is a complication associated with diabetes (e.g., diabetic retinopathy, diabetic nephropathy, kidney disease (e.g., renal failure, nephropathy and/or other diseases and disorders as described in the "Renal Disorders" section below), diabetic neuropathy, nerve disease and nerve damage (e.g., due to diabetic neuropathy), blood vessel blockage, heart disease, stroke, impotence (e.g., due to diabetic neuropathy or blood vessel

				<p>Exemplary assays that may be used or routinely modified to test for stimulation of insulin secretion (from pancreatic cells) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Shimizu, H., et al., Endocr J, 47(3):261-9 (2000); Salapatek, A.M., et al., Mol Endocrinol, 13(8):1305-17 (1999); Filipsson, K., et al., Ann N Y Acad Sci, 865:441-4 (1998); Olson, L.K., et al., J Biol Chem, 271(28):16544-52 (1996); and, Miraglia S et. al., Journal of Biomolecular Screening, 4:193-204 (1999), the contents of each of which is herein incorporated by reference in its entirety.</p> <p>Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include HIT-T15 Cells.</p>	<p>blockage), seizures, mental confusion, drowsiness, nonketotic hyperglycemic-hyperosmolar coma, cardiovascular disease (e.g., heart disease, atherosclerosis, microvascular disease, hypertension, stroke, and other diseases and disorders as described in the "Cardiovascular Disorders" section below), dyslipidemia, endocrine disorders (as described in the "Endocrine Disorders" section below), neuropathy, vision impairment (e.g., diabetic retinopathy and blindness), ulcers and impaired wound healing, and infection (e.g., infectious diseases and disorders as described in the "Infectious Diseases" section below, especially of the urinary tract and skin), carpal tunnel syndrome and Dupuytren's contracture). An additional highly preferred indication is obesity and/or complications associated with obesity. Additional highly</p>
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				<p>HITT15 are an adherent epithelial cell line established from Syrian hamster islet cells transformed with SV40. These cells express glucagon, somatostatin, and glucocorticoid receptors. The cells secrete insulin, which is stimulated by glucose and glucagon and suppressed by somatostatin or glucocorticoids. ATTC# CRL-1777 Refs: Lord and Ashcroft. Biochem. J. 219: 547-551; Santerre et al. Proc. Natl. Acad. Sci. USA 78: 4339-4343, 1981.</p>	<p>preferred indications include weight loss or alternatively, weight gain. Additional highly preferred indications are complications associated with insulin resistance.</p>
64	HFTBM50	368	<p>Production of IL-10 and activation of T-cells.</p>	<p>Assays for production of IL-10 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells. Exemplary assays that may be used or routinely modified to</p>	<p>Highly preferred indications include allergy and asthma. Additional highly preferred indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below),</p>

				<p>assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation and activation</p>	<p>immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response.</p>
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65	HFXDJ75	369	<p>Activation of transcription through API response element in immune cells (such as T-cells).</p>	<p>of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.</p>	<p>Assays for the activation of transcription through the API response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate growth and other cell functions. Exemplary assays for transcription through the API response element that may be used or routinely modified to test API-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene</p>	<p>Preferred indications include neoplastic diseases (e.g., as described below under "Hyperproliferative Disorders"), blood disorders (e.g., as described below under "Immune Activity", "Cardiovascular Disorders", and/or "Blood-Related Disorders"), and infection (e.g., an infectious disease as described below under "Infectious Disease"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Additional</p>
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				<p>66:1-10 (1988); Cullen and Malm, <i>Methods in Enzymol</i> 216:362-368 (1992); Henthorn et al., <i>Proc Natl Acad Sci USA</i> 85:6342-6346 (1988); Rellahan et al., <i>J Biol Chem</i> 272(49):30806-30811 (1997); Chang et al., <i>Mol Cell Biol</i> 18(9):4986-4993 (1998); and Fraser et al., <i>Eur J Immunol</i> 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. Human T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is an IL-2 and IL-4 responsive suspension-culture cell line.</p>	<p>highly preferred indications include inflammation and inflammatory disorders. Highly preferred indications also include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under “Hyperproliferative Disorders”). Highly preferred indications include neoplasms and cancers, such as, leukemia, lymphoma, prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver, and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include arthritis, asthma, AIDS, allergy, anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin’s disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt’s lymphoma,</p>
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65	HFXDJ75	369	Activation of transcription through CD28 response element in immune cells (such as T-cells).	Assays for the activation of transcription through the CD28 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate IL-2 expression in T cells. Exemplary assays for transcription through the CD28 response element that may be used or routinely modified to test CD28-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol	granulomatous disease, inflammatory bowel disease, sepsis, psoriasis, suppression of immune reactions to transplanted organs and tissues, endocarditis, meningitis, and Lyme Disease.
				<p>A highly preferred embodiment of the invention includes a method for stimulating T cell proliferation.</p> <p>An alternative highly preferred embodiment of the invention includes a method for inhibiting T cell proliferation.</p> <p>A highly preferred embodiment of the invention includes a method for activating T cells. An alternative highly preferred embodiment of the invention includes a method for inhibiting the activation of and/or inactivating T cells.</p> <p>A highly preferred embodiment of the invention includes a method for stimulating (e.g., increasing) IL-2 production. An alternative highly preferred embodiment</p>	

			<p>216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); McGuire and Iacobelli, J Immunol 159(3):1319-1327 (1997); Parra et al., J Immunol 166(4):2437-2443 (2001); and Butscher et al., J Biol Chem 3(1):552-560 (1998), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.</p>	<p>of the invention includes a method for inhibiting (e.g., reducing) IL-2 production. Additional highly preferred indications include inflammation and inflammatory disorders. Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Highly preferred indications include neoplastic diseases (e.g., melanoma, renal cell carcinoma, leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, for example, melanoma (e.g., metastatic melanoma), renal</p>
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					<p>cell carcinoma (e.g., metastatic renal cell carcinoma), leukemia, lymphoma (e.g., T cell lymphoma), and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. A highly preferred indication includes infection (e.g., AIDS, tuberculosis, infections associated with granulomatous disease, and osteoporosis, and/or as described below under "Infectious Disease"). A highly preferred indication is AIDS. Additional highly preferred indications include suppression of immune reactions to transplanted organs and/or tissues, uveitis, psoriasis, and tropical spastic paraparesis. Preferred indications include blood disorders (e.g., as described</p>
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					below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Preferred indications also include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, asthma and allergy.
65	HFXDJ75	369	Activation of transcription through NFKB response element in immune cells (such as T-cells).	Assays for the activation of transcription through the NFKB response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription factors and	Highly preferred indications include inflammation and inflammatory disorders. Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Highly preferred indications include autoimmune diseases

				<p>modulate expression of immunomodulatory genes. Exemplary assays for transcription through the NFKB response element that may be used or routinely modified to test NFKB-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Black et al., Virus Gnes 15(2):105-117 (1997); and Fraser et al., 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these</p>	<p>(e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), and immunodeficiencies (e.g., as described below). An additional highly preferred indication is infection (e.g., AIDS, and/or an infectious disease as described below under "Infectious Disease"). Highly preferred indications include neoplastic diseases (e.g., melanoma, leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, melanoma, renal cell carcinoma, leukemia, lymphoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for</p>
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				assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.	example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications also include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, suppression of immune reactions to transplanted organs, asthma and allergy.
66	HFXJU68	370	Activation of transcription through cAMP response element (CRE) in pre-adipocytes.	Assays for the activation of transcription through the cAMP response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to increase cAMP,	A highly preferred indication is obesity and/or complications associated with obesity. Additional highly preferred indications include weight loss or alternatively, weight gain. An additional highly preferred indication is diabetes mellitus. An additional highly preferred indication is a complication

				<p>regulate CREB transcription factors, and modulate expression of genes involved in a wide variety of cell functions. For example, a 3T3-L1/CRE reporter assay may be used to identify factors that activate the cAMP signaling pathway. CREB plays a major role in adipogenesis, and is involved in differentiation into adipocytes. CRE contains the binding sequence for the transcription factor CREB (CRE binding protein). Exemplary assays for transcription through the cAMP response element that may be used or routinely modified to test cAMP-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn</p>	<p>associated with diabetes (e.g., diabetic retinopathy, diabetic nephropathy, kidney disease (e.g., renal failure, nephropathy and/or other diseases and disorders as described in the "Renal Disorders" section below), diabetic neuropathy, nerve disease and nerve damage (e.g., due to diabetic neuropathy), blood vessel blockage, heart disease, stroke, impotence (e.g., due to diabetic neuropathy or blood vessel blockage), seizures, mental confusion, drowsiness, nonketotic hyperglycemic-hypermolar coma, cardiovascular disease (e.g., heart disease, atherosclerosis, microvascular disease, hypertension, stroke, and other diseases and disorders as described in the "Cardiovascular Disorders" section below), dyslipidemia, endocrine disorders (as described in the "Endocrine Disorders" section below),</p>
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				<p>et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Reusch et al., Mol Cell Biol 20(3):1008-1020 (2000); and Klemm et al., J Biol Chem 273:917-923 (1998), the contents of each of which are herein incorporated by reference in its entirety. Pre-adipocytes that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary mouse adipocyte cells that may be used according to these assays include 3T3-L1 cells. 3T3-L1 is an adherent mouse preadipocyte cell line that is a continuous substrain of 3T3 fibroblast cells developed through clonal isolation and undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation conditions known in the art.</p>	<p>neuropathy, vision impairment (e.g., diabetic retinopathy and blindness), ulcers and impaired wound healing, and infection (e.g., infectious diseases and disorders as described in the "Infectious Diseases" section below, especially of the urinary tract and skin), carpal tunnel syndrome and Dupuytren's contracture). Additional highly preferred indications are complications associated with insulin resistance.</p>
66	HFXJU68	370	Inhibition of squalene synthetase gene transcription.	Reporter Assay: construct contains regulatory and coding sequence of squalene	

			<p>synthetase, the first specific enzyme in the cholesterol biosynthetic pathway. See Jiang, et al., J. Biol. Chem. 268:12818-12824(1993), the contents of which are herein incorporated by reference in its entirety. Cells were treated with SID supernatants, and SEAP activity was measured after 72 hours. HepG2 is a human hepatocellular carcinoma cell line (ATCC HB-8065). See Knowles et al., Science. 209:497-9 (1980), the contents of which are herein incorporated by reference in its entirety.</p>	
66	HFXJU68	370	<p>Assays for production of IL-10 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells. Exemplary assays that may be</p>	<p>Highly preferred indications include allergy and asthma. Additional highly preferred indications include immune and hematopoietic disorders (e.g., as described below under "Immune Activity", and "Blood-Related Disorders"), autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis</p>

				<p>used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce</p>	<p>and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response.</p>
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				differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.	
67	HGBIB74	371	Activation of transcription through NFAT response element in immune cells (such as natural killer cells).	Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-	Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional highly preferred indications include inflammation and

				<p>response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Aramburu et al., J Exp Med 182(3):801-810 (1995); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Fraser et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J Biol Chem 268(19):14285-14293 (1993), the contents of each of which are herein incorporated by reference in its entirety. NK cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human NK cells that may be used according to these assays include the NK-YT cell line, which is a human</p>	<p>inflammatory disorders. An additional highly preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease"). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Preferred indications include neoplasms and cancers, such as, for example, leukemia, lymphoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications also include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple</p>
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					natural killer cell line with cytolytic and cytotoxic activity.	myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, asthma and allergy.
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				<p>that may be used or routinely modified to test SRE activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Benson et al., J Immunol 153(9):3862-3873 (1994); and Black et al., Virus Genes 12(2):105-117 (1997), the content of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary T cells that may be used according to these assays include the NK-YT cell line, which is a human natural killer cell line with cytolytic and cytotoxic activity.</p>	<p>(e.g., rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional highly preferred indications include inflammation and inflammatory disorders, and treating joint damage in patients with rheumatoid arthritis. An additional highly preferred indication is sepsis. Highly preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Additionally, highly preferred indications include neoplasms and cancers, such as, for example, leukemia, lymphoma, melanoma, glioma (e.g., malignant glioma), solid tumors, and prostate, breast,</p>
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					lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, cardiac reperfusion injury, and asthma and allergy. An additional preferred indication is infection (e.g., an infectious
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67	HGBIB74	371	Activation of transcription through GAS response element in immune cells (such as T-cells).	Assays for the activation of transcription through the Gamma Interferon Activation Site (GAS) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate STAT transcription factors and modulate gene expression involved in a wide variety of cell functions. Exemplary assays for transcription through the GAS response element that may be used or routinely modified to test GAS-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn	disease as described below under "Infectious Disease").
				Highly preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, for example, leukemia, lymphoma (e.g., T cell lymphoma, Burkitt's lymphoma, non-Hodgkins lymphoma, Hodgkin's disease), melanoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described	

				<p>et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Matikainen et al., Blood 93(6):1980-1991 (1999); and Henttinen et al., J Immunol 155(10):4582-4587 (1995), the contents of each of which are herein incorporated by reference in its entirety. Exemplary human T cells, such as the SUPT cell line, that may be used according to these assays are publicly available (e.g., through the ATCC).</p>	<p>below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional preferred indications include inflammation and inflammatory disorders. Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"), and infection (e.g., viral infections, tuberculosis, infections associated with chronic granulomatous disease and malignant osteoporosis, and/or an infectious disease as described below under "Infectious Disease"). An additional preferred indication is idiopathic pulmonary fibrosis. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, acute lymphocytic anemia</p>
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68	HHEMA75	372	<p>Activation of transcription through cAMP response element in immune cells (such as T-cells).</p>	<p>Assays for the activation of transcription through the cAMP response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to increase cAMP, bind to CREB transcription factor, and modulate expression of genes involved in a wide variety of cell functions. Exemplary assays for transcription through the cAMP response element that</p>	<p>(ALL), plasmacytomas, multiple myeloma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, and asthma and allergy.</p>	<p>Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"), and infection (e.g., an infectious disease as described below under "Infectious Disease"). Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below),</p>
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			<p>may be used or routinely modified to test cAMP-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Black et al., Virus Genes 15(2):105-117 (1997); and Belkowski et al., J Immunol 161(2):659-665 (1998), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC).</p> <p>Exemplary human T cells that may be used according to these assays include the JURKAT cell line, which is a suspension culture of leukemia cells that produce IL-2 when stimulated.</p>	<p>boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional preferred indications include inflammation and inflammatory disorders. Highly preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, leukemia, lymphoma (e.g., T cell lymphoma, Burkitt's lymphoma, non-Hodgkins lymphoma, Hodgkin's disease), melanoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include</p>
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					<p>anemia, pancytopenia, leukopenia, thrombocytopenia, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, and asthma and allergy.</p>
68	HHEMA75	372	<p>Activation of transcription through NFAT response element in immune cells (such as natural killer cells).</p>	<p>Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in</p>	<p>Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T</p>

			<p>immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Aramburu et al., J Exp Med 182(3):801-810 (1995); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Fraser et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J Biol Chem 268(19):14285-14293 (1993), the contents of each of which are herein incorporated by reference in its entirety. NK cells that may be used according to these assays are</p>	<p>cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional highly preferred indications include inflammation and inflammatory disorders. An additional highly preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease"). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Preferred indications include neoplasms and cancers, such as, for example, leukemia, lymphoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia.</p>
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				publicly available (e.g., through the ATCC). Exemplary human NK cells that may be used according to these assays include the NK-YT cell line, which is a human natural killer cell line with cytolytic and cytotoxic activity.	Preferred indications also include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, asthma and allergy.
68	HHEMA75	372	Activation of transcription through AP1 response element in immune cells (such as T-cells).	Assays for the activation of transcription through the AP1 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate growth and other cell functions. Exemplary assays for	Preferred indications include neoplastic diseases (e.g., as described below under "Hyperproliferative Disorders"), blood disorders (e.g., as described below under "Immune Activity", "Cardiovascular Disorders", and/or "Blood-Related Disorders"), and infection (e.g., an infectious disease as described below under

				<p>transcription through the AP1 response element that may be used or routinely modified to test AP1-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1988); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Rellahan et al., J Biol Chem 272(49):30806-30811 (1997); Chang et al., Mol Cell Biol 18(9):4986-4993 (1998); and Fraser et al., Eur J Immunol 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. Human T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell</p>	<p>“Infectious Disease”). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below) and immunodeficiencies (e.g., as described below). Additional highly preferred indications include inflammation and inflammatory disorders. Highly preferred indications also include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under “Hyperproliferative Disorders”). Highly preferred indications include neoplasms and cancers, such as, leukemia, lymphoma, prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver, and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia,</p>
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				line, which is an IL-2 and IL-4 responsive suspension-culture cell line.	metaplasia, and/or dysplasia. Preferred indications include arthritis, asthma, AIDS, allergy, anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, granulomatous disease, inflammatory bowel disease, sepsis, psoriasis, suppression of immune reactions to transplanted organs and tissues, endocarditis, meningitis, and Lyme Disease.
68	HHEMA75	372	Activation of transcription through CD28 response element in immune cells (such as T-cells).	Assays for the activation of transcription through the CD28 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate IL-2 expression in T cells. Exemplary assays for transcription through the CD28 response element that may be	<p>A highly preferred embodiment of the invention includes a method for stimulating T cell proliferation. An alternative highly preferred embodiment of the invention includes a method for inhibiting T cell proliferation. A highly preferred embodiment of the invention includes a method for activating T cells. An alternative highly preferred embodiment of the invention</p>

			<p>used or routinely modified to test CD28-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); McGuire and Iacobelli, J Immunol 159(3):1319-1327 (1997); Parra et al., J Immunol 166(4):2437-2443 (2001); and Butscher et al., J Biol Chem 3(1):552-560 (1998), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC).</p> <p>Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4</p>	<p>includes a method for inhibiting the activation of and/or inactivating T cells. A highly preferred embodiment of the invention includes a method for stimulating (e.g., increasing) IL-2 production. An alternative highly preferred embodiment of the invention includes a method for inhibiting (e.g., reducing) IL-2 production. Additional highly preferred indications include inflammation and inflammatory disorders. Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Highly preferred indications include neoplastic diseases (e.g., melanoma, renal</p>
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				responsive T cells.	<p>cell carcinoma, leukemia, lymphoma, and/or as described below under</p> <p>“Hyperproliferative Disorders”). Highly preferred indications include neoplasms and cancers, such as, for example, melanoma (e.g., metastatic melanoma), renal cell carcinoma (e.g., metastatic renal cell carcinoma), leukemia, lymphoma (e.g., T cell lymphoma), and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. A highly preferred indication includes infection (e.g., AIDS, tuberculosis, infections associated with granulomatous disease, and osteoporosis, and/or as described below under “Infectious Disease”). A highly preferred indication is</p>
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					<p>AIDS. Additional highly preferred indications include suppression of immune reactions to transplanted organs and/or tissues, uveitis, psoriasis, and tropical spastic paraparesis. Preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Preferred indications also include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma, arthritis, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, asthma and allergy.</p>
68	HHEMA75	372	Activation of transcription	Assays for the activation of transcription through the	Highly preferred indications include neoplastic diseases

			through GAS response element in immune cells (such as T-cells).	<p>Gamma Interferon Activation Site (GAS) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate STAT transcription factors and modulate gene expression involved in a wide variety of cell functions. Exemplary assays for transcription through the GAS response element that may be used or routinely modified to test GAS-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Matikainen et al., Blood 93(6):1980-1991 (1999); and</p>	<p>(e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, for example, leukemia, lymphoma (e.g., T cell lymphoma, Burkitt's lymphoma, non-Hodgkins lymphoma, Hodgkin's disease), melanoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and</p>
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				<p>Henttinen et al., J Immunol 155(10):4582-4587 (1995), the contents of each of which are herein incorporated by reference in its entirety. Exemplary human T cells, such as the SUPT cell line, that may be used according to these assays are publicly available (e.g., through the ATCC).</p>	<p>suppressing a T cell-mediated immune response. Additional preferred indications include inflammation and inflammatory disorders. Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"), and infection (e.g., viral infections, tuberculosis, infections associated with chronic granulomatous disease and malignant osteoporosis, and/or an infectious disease as described below under "Infectious Disease"). An additional preferred indication is idiopathic pulmonary fibrosis. Preferred indications include anemia, pancytopenia, leukopenia, thrombocytopenia, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, arthritis, AIDS, granulomatous disease, inflammatory bowel disease,</p>
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68	HHEMA75	372	Activation of transcription through NFAT response element in immune cells (such as T-cells).	Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of	sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, and asthma and allergy.
				Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), immunodeficiencies (e.g., as described below), boosting a T cell-mediated immune response, and suppressing a T cell-mediated immune response. Additional highly preferred indications include inflammation and inflammatory disorders. An	

				<p>polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Serfling et al., Biochim Biophys Acta 1498(1):1-18 (2000); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Fraser et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J Biol Chem 268(19):14285-14293 (1993), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4</p>	<p>additional highly preferred indication is infection (e.g., an infectious disease as described below under "Infectious Disease"). Preferred indications include neoplastic diseases (e.g., leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Preferred indications include neoplasms and cancers, such as, for example, leukemia, lymphoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications also include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple myeloma, Burkitt's lymphoma,</p>
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					responsive T cells.	arthritis, AIDS, granulomatous disease, inflammatory bowel disease, sepsis, neutropenia, neutrophilia, psoriasis, suppression of immune reactions to transplanted organs and tissues, hemophilia, hypercoagulation, diabetes mellitus, endocarditis, meningitis, Lyme Disease, asthma and allergy.
					Assays for the activation of transcription through the NFKB response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription through the NFKB response element that may be used or routinely modified to test NFKB-response element activity of	Highly preferred indications include inflammation and inflammatory disorders. Highly preferred indications include blood disorders (e.g., as described below under "Immune Activity", "Blood-Related Disorders", and/or "Cardiovascular Disorders"). Highly preferred indications include autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis and/or as described below), and immunodeficiencies (e.g., as described below). An additional highly preferred indication is infection (e.g.,
					Activation of transcription through NFKB response element in immune cells (such as T-cells).	
					372	
					HHEMA75	
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				<p>polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Black et al., Virus Gnes 15(2):105-117 (1997); and Fraser et al., 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.</p>	<p>AIDS, and/or an infectious disease as described below under "Infectious Disease"). Highly preferred indications include neoplastic diseases (e.g., melanoma, leukemia, lymphoma, and/or as described below under "Hyperproliferative Disorders"). Highly preferred indications include neoplasms and cancers, such as, melanoma, renal cell carcinoma, leukemia, lymphoma, and prostate, breast, lung, colon, pancreatic, esophageal, stomach, brain, liver and urinary cancer. Other preferred indications include benign dysproliferative disorders and pre-neoplastic conditions, such as, for example, hyperplasia, metaplasia, and/or dysplasia. Preferred indications also include anemia, pancytopenia, leukopenia, thrombocytopenia, Hodgkin's disease, acute lymphocytic anemia (ALL), plasmacytomas, multiple</p>
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